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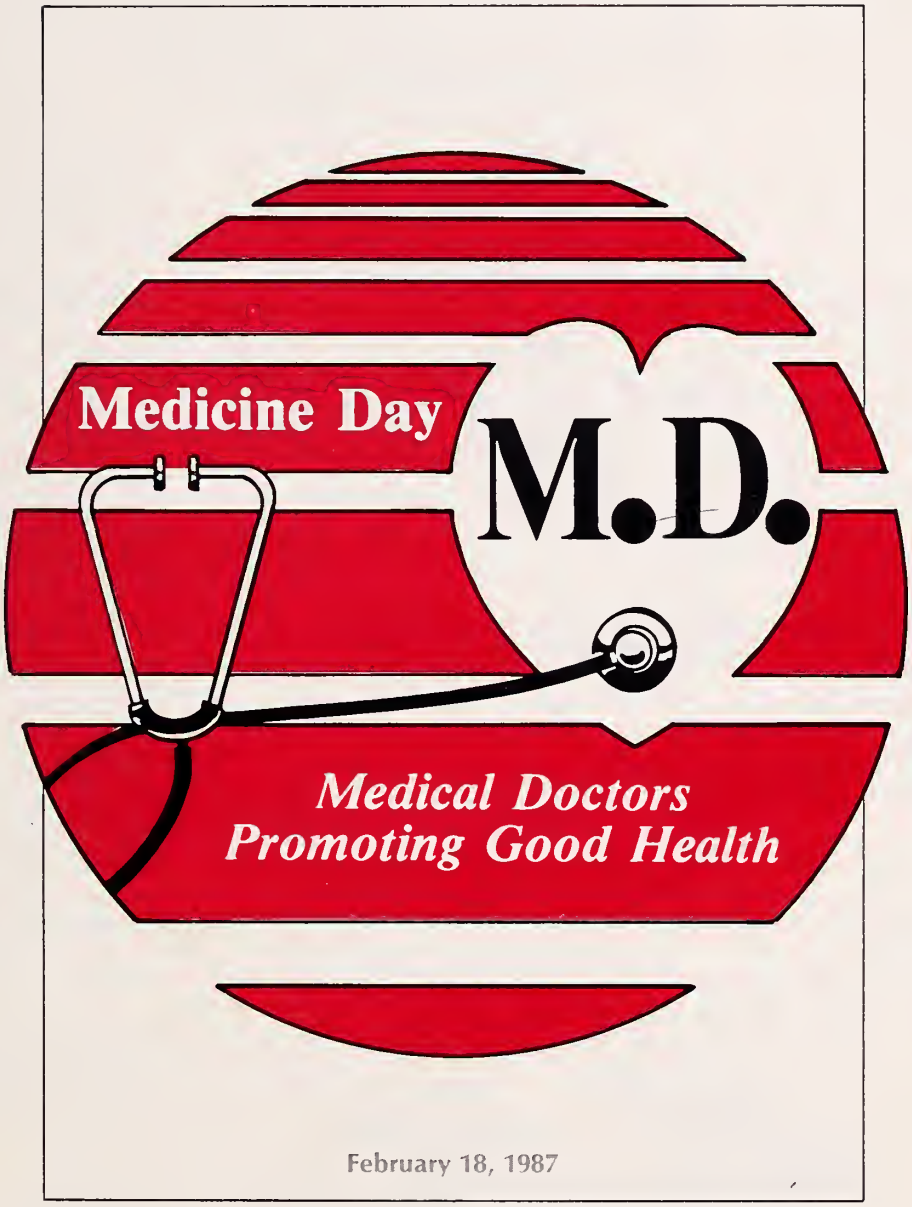
OKLAHOMA STATE MEDICAL ASSOCIATION
JANUARY 1987

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# JOURNAL

## OKLAHOMA STATE MEDICAL ASSOCIATION

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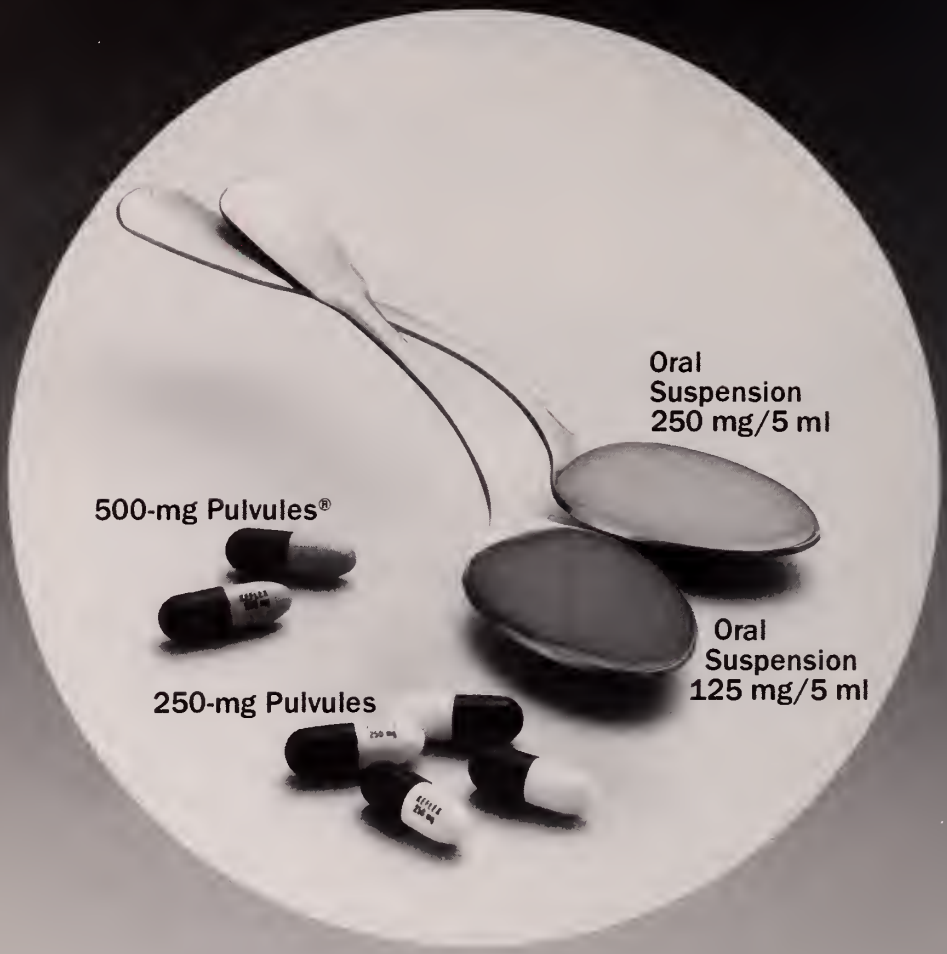


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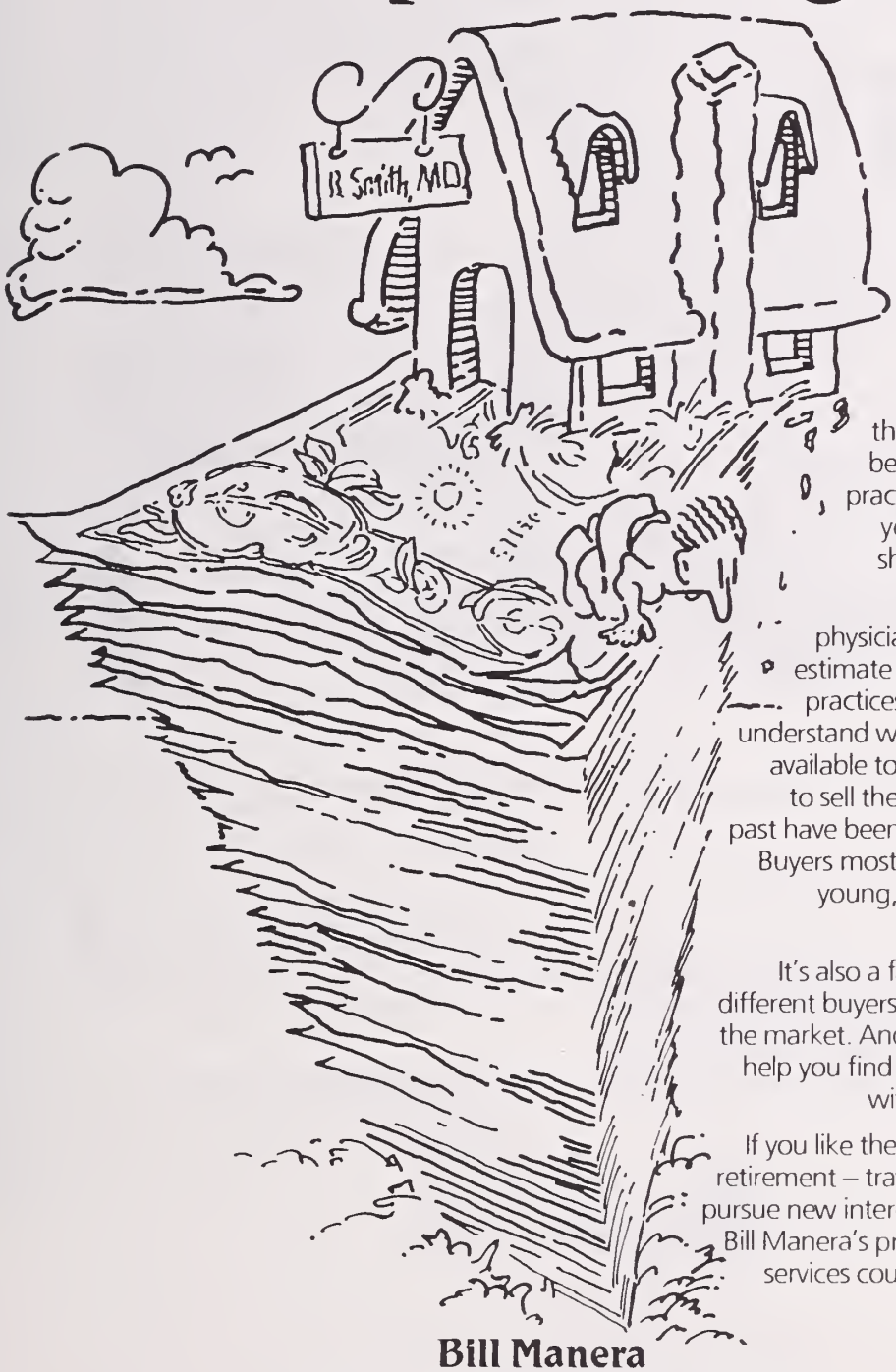


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# Ah — The Golden Years

Honestly, I've done my best to start the new year on a positive if not altogether optimistic note. My best turned out to be sorry. In an exhausting effort, I combed newspapers, journals, and official federal and state publications, searching for the sunbeams that would portend the dawn of the golden age of medicine. Here are some samples of my discoveries:

If the physician's average actual charge or MAAC for a service during the previous year (whichever is lower) is less than 115 percent of the current prevailing charge for nonparticipants, the physician's MAAC for the current year is the greater of 101 percent of the MAAC for the previous year *or* the MAAC for the previous year plus a fraction of the difference between the MAAC for the previous year and 115 percent of the prevailing charge. For 1987, this fraction is one-quarter; for 1988, one-third; for 1989, one-half; and for any subsequent year, one.

For purposes of determining a nonparticipating physician's MAAC for 1987, the previous year's (1986) MAAC is deemed by law to be the physician's charge during the April-June 1984 base period, if the physician had charges during that period. (This rule applies for all 1987 nonparticipating physicians, regardless of their prior participation status.)[1]

Under Public Law 99-509, the Omnibus Budget Reconciliation Act of 1986, there continue to be limits on the charges to Medicare beneficiaries which may be made by nonparticipating physicians. Also, as in the past, if a nonparticipating physician violates those limits, he or she may be subject to sanctions such as exclusion from the Medicare program and/or the payment of civil money penalties. In addition, charges in excess of the charge limit continue to be excluded when computing a nonparticipating physician's customary charge. These charge limits continue until 1990 or until one year after development of a national relative value scale for making Medicare payment for physicians' services, whichever comes first.[2]

The Department has compiled and published a comprehensive schedule which contains all CPT, HCFA and Aetna Codes, as well as the DHS Specific Codes, and the rate of reimbursement for each.[3] [Attached are 50 pages of codes and reimbursement amounts.]

Public Law 99-177, The Balanced Budget and Emergency Deficit Control Act of 1985, commonly known as Gramm-Rudman-Hollings Bill, requires that payment for all Medicare services be reduced by one percent for services rendered March 1, 1986 through September 30, 1986.[4]

Physician Liability on PRO Denials For all admissions on and after January 1, 1987 physicians will be required to refund any Medicaid payments made to them for services provided Medicaid recipients when it has been determined that such admissions or any part of the hospital stay did not meet the Medicaid necessity criteria applied by the PRO.[5]

MDs must calculate Medicare pay data[6]

HCFA to use law to delay claims[7]

Med school admissions down for 5th year[8]

Johns Hopkins center adapting model for 'barefoot doctors'[9]

Almost 60% of physicians and more than 77% of medical students surveyed in a Massachusetts study claim to have used psychoactive drugs (not alcohol) for self-treatment, to get high, or to maintain their work performance.[10]

It must be that I am reading too much or reading all the wrong things. Perhaps I am misinterpreting the meaning or significance of all this information.

On the other hand, I could be having a problem with semantics. After all, there is solid gold, gold plate, gold filled, and fool's gold.

—*MRJ*


1. Fact Sheet, distributed by Aetna as Medicare intermediary, p 3.
2. Ibid, p 3.
3. Letter to providers, Oklahoma Dept of Human Services, Oct 20, 1986, p 1.
4. *Medicare News*, Vol 14, No 2, Dec 1986.
5. Lettter to doctors. Okla Dept of Human Services, Nov 18, 1986, p 1.
6. Headline, *American Medical News*, Nov 28, 1986, p 3.
7. Headline, *American Medical News*, Dec 5, 1986, p 1.
8. Ibid, p 14.
9. Ibid, p 14.
10. *US Journal of Drug and Alcohol Dependence*, Vol 10, No 11, Nov 1986, p 1.

Dear Fellow Physicians:

As onerous as the regulations, restrictions, and controls on medicine, placed on us by the government through Medicare, may be, a specter even worse is arising. I'm referring to the so-called private insurance companies.

As competition for the patient's premium dollar increases, these companies are attempting to withhold many services from patients in order to save money and, therefore, to compete more efficiently in the marketplace.

The confusions of precertification, the threat implied with second opinions, the demand for same-day admissions for elective surgery, the restrictions placed on length of hospitalization, and the urgency toward outpatient surgery for most everything, are all attempts to save money, often while jeopardizing the patient's safety and well-being with little regard to the opinions of the attending physician.

At least the government can be reasoned with and often will alter their regulations. Their goal is still service to the people, and eventually they must answer to the public through our elected officials.

On the other hand, private insurance is only interested in the bottom-line dollar; there is no drive at all for the public's health and safety. Decisions are almost invariably made based exclusively on economic profitability and often by a rather disinterested party.

It is ironic that we've reached a point where the government is much more concerned with sound, safe health practices than some of our "friends" in private industry.

Norman L. Dunitz, MD

# Thyrotoxic Hypokalemic Periodic Paralysis: Report of Four Cases and Review of the Literature

(First of two parts)

THOMAS D. TINKER, MD, and JERRY B. VANNATTA, MD

**Hyperthyroidism is associated with several neuromuscular disorders, one of which is periodic paralysis. Hypokalemia almost always accompanies the dramatic paralytic episodes which characterize this rare disorder, hence the name thyrotoxic hypokalemic periodic paralysis (TPP). Presented are four cases, including the first reported in a Vietnamese male and the first associated with lymphocytic thyroiditis. One (case 3) is of undetermined cause. A review of the literature is included, as well as a thorough discussion of clinical findings and pathophysiologic mechanisms. A comparison between TPP and familial periodic paralysis is made.**

Hyperthyroidism is associated with a number of muscular disorders including acute and chronic thyrotoxic myopathies, exophthalmic ophthalmoplegia, myasthenia gravis, and periodic paralysis.[1] Among these, periodic paralysis is probably the rarest complication of hyperthyroidism. Because hypokalemia commonly accompanies the paralytic attacks in these hyperthyroid patients, the disease is also known as thyrotoxic hypokalemic periodic paralysis (TPP). This paper presents the literature's first report in a Vietnamese male, the fourth and fifth in American Indian males, and the thirtieth in a Caucasian male.

From the University of Oklahoma College of Medicine and Presbyterian Hospital, Oklahoma City, Oklahoma.

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**Case 1** A 22-year-old Vietnamese man presented with a 10-day history of generalized weakness that intermittently impaired walking. He acknowledged insomnia, increased irritability, heat intolerance, palpitations, hand tremors, and a five-pound weight loss in the preceding two weeks. On the morning of admission, he was awakened from sleep by palpitations and severe bilateral leg cramps. He denied profuse vomiting and diarrhea, licorice or thyroid medication intake, and the use of illicit drugs, diuretics, and laxatives. There was no family history of a similar disorder.

On examination his pulse rate was 120 beats per minute and blood pressure was 120/76 mm Hg. Lid lag and prominent stare were noted. The patient's thyroid was mildly enlarged and asymmetric. Marked generalized weakness was present and the lower extremities were bilaterally tender to palpation. Except for a fine tremor of the hands and

**The patient's muscular weakness resolved with oral potassium supplementation . . .**

hypoactive deep tendon reflexes, the results of the remainder of the neurologic examination, including sensory and mental status, were within normal limits. On admission the patient's serum potassium, phosphorus, and magnesium levels were 1.4 mEq/1 (3.5-5.0 mEq/1), 1.7 mg/dl (2.5-4.5 mg/dl), and 1.6 mg/dl (1.8-2.4 mg/dl), respectively. The electrocardiogram revealed first-degree atrioventricular block with nonspecific ST junction depression.

Administration of intravenous fluids with potassium supplementation (10mEq/hr) was initiated. Within 12 hours the patient's serum potassium rose to 4.7 mEq/1 and subsequently remained stable despite the absence of further potassium administration during the hospital course.

Thyroid function studies revealed a tri-iodine resin uptake ($T_3RU$) of 44% (25%-35%), serum thyroxine by RIA ($T_4$) of 18.2 ug/dl (4.5-12.0 ug/dl), and free thyroxine index (FTI) of 8.0 (1.1-4.2). Relief of anxiety and sweating, as well as control of tachycardia, was achieved with nadolol. A subsequent ECG showed resolution of the first-degree AV block and ST junction depression. Twenty-four-hour radioactive iodine uptake (24 hr RAIU) was 3% (10%-35%), excluding Graves disease as the cause of hyperthyroidism. Antithyroid microsomal and thyroglobulin antibodies were negative. Needle aspiration biopsy of the thyroid gland yielded thyroid follicular cells and sheets of lymphocytes. The diagnosis of lymphocytic thyroiditis was made. When discharged from the hospital, the patient was on a regimen of nadolol 160 mg twice daily and was instructed to decrease the dosage by half each time his pulse rate declined to 70 beats per minute. At the time of discharge the patient's serum potassium, phosphorus, and magnesium levels had risen to 4.3 mEq/1, 5.0 mg/dl, and 2.2 mg/dl, respectively.

One month later, the patient was clinically euthyroid and had discontinued nadolol. Other than a single episode of lower extremity paralysis shortly after his discharge, which resolved spontaneously within five hours, the patient had been asymptomatic and had returned to work.

**Case 2** The patient, a 20-year-old American Indian man, felt well until the morning of his admission, when he awoke and had difficulty getting out of bed. He had pain in his quadriceps, and standing was difficult. A similar episode of muscle weakness had occurred one year prior to his admission and had resolved spontaneously within 24-36 hours. He denied heat intolerance, diarrhea, and history of drug use, but did admit to a 100-lb weight loss without effort over the preceding year. There was no family history of periodic paralysis or thyroid disorder.

**Table 1. — Classification of Periodic Paralysis**

I. Primary periodic paralysis
   A. Familial hypokalemic periodic paralysis
   B. Hyperkalemic paralysis with or without myotonia
   C. Normokalemic periodic paralysis
   D. Paramyotonia congenita with myotonia and weakness on exposure to cold

II. Secondary periodic paralysis
   A. Thyrotoxic hypokalemic periodic paralysis
   B. Paralysis associated with other endocrinologic causes of hypokalemia, ie:
      1. Mineralocorticoid excess
      2. Renal tubular acidosis
      3. Diabetic ketoacidosis
      4. Licorice intoxication

Physical examination revealed the patient's blood pressure was 160/60 mm Hg and pulse rate was 134 beats per minute. He appeared anxious and was unable to sit or stand unaided at the time of admission. His skin was warm without excessive moisture. No lid lag or proptosis was noted. The patient's neck was supple with diffuse thyromegaly, and bruits were heard over the right lobe and isthmus of his thyroid. No nodules were palpated. Neurologic examination revealed proximal muscle paresis primarily involving the quadriceps, biceps, and triceps. Deep tendon reflexes and sensory examinations showed normal reactions, and the remainder of the findings on physical examination were within normal limits.

On admission the patient's serum potassium and creatine phosphokinase (CPK) levels were 1.9 mEq/liter and 600 IU/liter, (16-160 IU/liter), respectively. Thyroid function tests revealed $T_3RU$ of 42% (23%-35%), serum $T_4$ of 14.8 mg/dl (4.5-12.0 mg/dl), and FTI of 5.3 (1.1-4.2). Thyroid uptake and scan on the fourth hospital day showed a 24-hour uptake of 39% (10%-35%) and a calculated thyroid gland weight of 46 grams, all consistent with the diagnosis of Graves disease.

The patient's muscular weakness resolved with oral potassium supplementation and the serum potassium levels rose to normal ranges. The diagnosis of hypokalemic periodic paralysis associated with thyrotoxicosis was made and the patient was treated with 11.8 mCi of $^{131}I$. After four days without adverse reactions to the treatment, potassium therapy was stopped and the patient was

discharged from the hospital and advised to take 10 mg of propranolol four times daily. Serum potassium on discharge was 4.3 mEq/liter. At one month post-discharge, he was symptom-free and subsequently lost to follow-up.

**Case 3** A 26-year-old American Indian man was admitted following three days of intermittent bilateral lower extremity weakness. His first "spell" had occurred suddenly approximately seven years earlier while he was walking down a staircase. Since that time similar episodes had recurred, usually about five times a year. The spells typically resolved spontaneously after several hours, often waxing and waning in their severity. Improvement with exercise occasionally was obtained. The most marked weakness was noted to occur after alcohol intake, during the wintertime, and in the morning, especially after vigorous exercise the preceding day.

Three days prior to admission, over a period of several hours, the patient experienced the onset of bilateral lower extremity weakness. The weakness was intermittent over the next 72 hours, occasionally becoming severe enough to impair walking. On the evening prior to admission, the patient noted weakness of both legs, both arms, and his abdominal muscles. He denied pain, circumoral paresthesias, dysphagia, dysarthria, and visual changes. No recent history of weight loss, diarrhea, excessive sweating, emotional lability, heat intolerance, or palpitations was reported. The patient denied the use of drugs, including thyroid medication, and his family history was negative for a similar disorder.

**The spells [of weakness] typically resolved spontaneously after several hours . . .**

Physical examination revealed his blood pressure to be 102/65 mm Hg and pulse rate to be 65 beats per minute. His neck was supple, and the possibility of a palpated mass in the right lobe of his thyroid was noted. A soft, Grade II/VI systolic murmur without gallop was present on auscultation along the left sternal border. The patient's head, chest, and abdomen were normal. Neurologic examination revealed hyperactive deep tendon reflexes and mild weakness of wrist and knee extensors. The remainder of the findings on examination, including mental status, cranial nerve, and sensory and cerebellar functions, were normal.

On admission, the patient's serum potassium, CPK, and calcium levels were 2.2. mEq/liter, 244 IU/liter, and 9.7 mg/dl, respectively. Thyroid function tests revealed $T_3RU$ of 39%, serum $T_4$ of 14.8 mg/dl, and FTI of 5.7. Intravenous potassium supplementation was initiated but was discontinued six hours later because the serum potassium had risen to 5.2 mEq/liter. A glucose infusion test with ECG and EMG monitoring was performed. Within 30 minutes of starting the infusion (D50 at 7 ml per minute), flattening of T waves was noted on the ECG. Weakness of the right hip flexor muscles was noted at 60 minutes, after the patient had received 10 units of regular insulin subcutaneously. At the test's conclusion (90 minutes), the patient had bilateral lower extremity weakness, primarily involving the hip flexors, and decreased amplitude of EMG potentials. Reflexes were preserved, and no arrhythmias were observed. The patient's 90-minute serum potassium was 2.5 mEq/liter. Muscular weakness and T wave flattening on the ECG subsequently resolved after 60 mEq of potassium chloride was given orally.

Because all indices of thyroid function were elevated, the patient was hypokalemic, and a provocative test was confirmative; the diagnosis of hyrotoxic hypokalemic periodic paralysis was made. The patient left the hospital against medical advice prior to the elucidation of the exact cause of thyrotoxicosis and was lost to follow-up.

**Case 4** The patient, a 29-year-old white man, was admitted to the hospital for evaluation of recurrent episodes of paralysis, affecting his upper and lower extremities without involvement of muscles of respiration, deglutition, extraocular movements, or sphincter control.

Four months prior to admission, while at work, the patient first noted "muscle soreness and heaviness," requiring that he "pick up (his) legs to climb a ladder." This resolved spontaneously in three days. One month later, after a large carbohydrate meal, the patient was unable to rise from a couch where he had lain for several hours watching television. He was taken to the local emergency room where his serum potassium and phosphorus levels

were determined to be 1.7 mEq/liter and 1.6 mg/dl, respectively. He was admitted to the hospital and treated with intravenous potassium chloride. Six hours later he was able to move without impairment. His serum potassium level was 5.1 mEq/liter when he was discharged and was 4.8 mEq/liter one week later.

Subsequently, one evening after an especially hard day at work, the patient found he was unable to support his weight upon standing. Later that same night he could not raise his legs against gravity, but his upper extremity muscles only ached. Once again he was taken to the local emergency room because of the paralysis. While in the emergency room he was found to have upper extremity proximal muscle weakness and lower extremity flaccid paresis. Deep tendon reflexes were hypoactive in the upper and absent in the lower extremities. No sensory abnormalities were noted. The patient's serum potassium and phosphorus levels were 2.4 mEq/liter and 1.5 mg/dl, respectively. CPK was 220 IU/liter. Weakness and paralysis again resolved following intravenous potassium chloride administration. During hospitalization, the following studies were performed and revealed no abnormalities: chest x-ray, IVP, barium enema, and upper GI series. A thyroid profile, however, revealed $T_3$RU of 40% (23%-32%), serum $T_4$ of 16 ug/dl (5-12 ug/dl), and FTI of 6.85 (1.25-4.20). At this point the patient was transferred to a tertiary care hospital.

**Musgrave is attributed with describing the first case of periodic paralysis in 1727.**

On the date of admission, the patient complained of palpitations and expressed a fear of dying. When questioned, he acknowledged a 50-pound weight loss during the preceding summer, which he attributed to voluntary dieting. Also acknowledged were several months of heat intolerance, excessive sweating, emotional lability, palpitations and, most recently, diarrhea. He was taking oral potassium supplements and Aldoril for hypertension. The potassium supplements and Aldoril were presumbly prescribed because of previous elevations of blood pressure and recurrent bouts of hypokalemia. His family history was negative for periodic paralysis but was positive for thyrotoxicosis in his mother and two sisters.

On examination the patient's blood pressure was found to be 140/80 mm Hg and his pulse rate was 100 beats per minute. Extraocular muscle function was intact and neither proptosis nor lid lag was noted. The patient's neck was supple, and his thyroid was diffusely enlarged without nodules or bruits. His heart had a regular rhythm without gallop. Neurologic examination revealed a mild tremor of out-stretched hands and weakness of all skeletal muscle groups in the arms and legs. Deep tendon reflexes were normal bilaterally. The remainder of the examination revealed nothing abnormal. At the time the patient was admitted, results of all laboratory tests were within normal limits except for the thyroid profile, which revealed $T_3$RU of 57% (35%-45%), serum $T_4$ of 16.3 ug/dl (6.0-12.0 ug/dl), and FTI of 9.3 (2.3-4.9). Thyroid uptake and scan revealed a 24-hour RAIU of 68% (5%-30%) with diffuse toxic goiter. The diagnosis of hypokalemic periodic paralysis associated with Graves disease was made. The patient subsequently was given 10 mCi of $^{131}$I and, after three days of observation, was discharged. His resting pulse rate was 88 per minute. He was advised to take propranolol, 40 mg QID, and the Aldoril was discontinued. Oral supplementation with potassium chloride, 20 mEq KCL, po BID, was also prescribed. At one week follow-up, the patient complained of muscle soreness but denied further episodes of weakness or paralysis. Overall he was much improved.

## History

Thyrotoxic periodic paralysis and familial periodic paralysis (Table 1) historically have been considered as distinct entities, but the separation between the two has always been less than clear. The following historic account of hypokalemic periodic paralysis (HPP) incorporates significant dates in the development of knowledge of both thyrotoxic and familial periodic paralysis.

Musgrave is attributed with describing the first case of periodic paralysis in 1727.[2] Shakhnovitch (1882), reporting the first case of familial periodic paralysis, described a 44-year-old man with periodic paralysis whose father had died with a similar illness.[2] In 1885 Westphal discovered a complete loss of muscular contractility to electrical stimulation during attacks.[2] Goldflam's observations in 1890 of the pathologic changes in affected muscles (ie, vacuolation and splitting of Cohnheim's fields) are

still accurate.[2] In 1902, Rosenfeld first reported, from Germany, an association between periodic paralysis and exophthalmic goiter.[2] Almost 30 years later, Dunlap and Kepler presented, in the English literature,[3] four patients with the same clinical findings.

In 1908, Kramer discovered that a high carbohydrate diet could induce paralytic attacks.[2] Shinosaki, in 1926, used glucose experimentally to induce attacks and reported that giving thyroid preparations to patients with TPP precipitated attacks.[4] Biemond and Daniels (1934), in a patient with familial periodic paralysis (FPP), first described the drop in serum potassium levels during attacks.[2] This finding, ie, hypokalemia during paralytic episodes, was confirmed in reports by Aitken et al, Allott and McArdle, Gammet et al, and Ferrebee et al by 1940.[2] Aitken et al had successfully used insulin to precipitate attacks in 1937.[2]

Talbott, in a classical review in 1941, summarized the clinical features of hypokalemic periodic paralysis.[5] In 1948, Gass et al,[6] showed that potassium is not lost in the urine or stool during attacks and suggested that potassium might become concentrated in muscles during paralysis. Grob et al and Zierler with Andres independently demonstrated (1957) a large uptake of potassium by skeletal muscle during paralytic attacks.[2] In the same year, Okinaka reported the incidence of TPP among thyrotoxic Japanese to be 1.9%.[7]

Engle's 1961 survey of the world literature on TPP produced 228 cases, 90% of which were reported in Japan.[8] Between 1966 and 1973, numerous metabolic and ultrastructural studies were undertaken to elucidate the biochemical aberrations responsible for and the histopathologic changes observed during the paralytic attacks characteristic of TPP.[9-18] The pathogenesis remains unknown, yet much has been learned about TPP since Rosenfeld's initial report more than eighty years ago.

**Note:** Part 2 of this article will appear in the February 1987 issue of the JOURNAL.

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# Phone Transmission of Fetal Heart Rate (FHR) Tracings in a Rural Setting

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**The use of a telemetry system permits small obstetrical units to have access to a technique often denied to them because of limited manpower and/or few deliveries.**

During the past decade, electronic fetal heart monitoring has become an integral component of the intrapartum, as well as antepartum, fetal surveillance. Fetal heart rate patterns, however, are occasionally difficult to interpret.

This problem is particularly troublesome in rural settings when it is essential to discriminate between normal and abnormal FHR patterns that require timely medical intervention.

We have previously described a telemetry system using transtelephonic transmission of fetal monitor data.[1] This system allows the obstetrical staffs in selected rural Oklahoma hospitals to obtain immediate consultation from our center. Results of our initial experience with this system are herein reported.

## Material and Methods

The characteristics of the transmission system have been described elsewhere.[1] The transmitting unit at the local hospital is connected to a telephone line and a fetal heart rate monitor.

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The circuitry includes a modulator, a demodulator, and an auto-answer unit. All transmissions are on-line and are implemented upon the request of the attending physician at the referring hospital. When a request for monitoring is received at the Eastern Oklahoma Perinatal Center (EOPC), pertinent clinical and obstetrical information is obtained and logged. Then a phone line is connected to a central display console and the data transmission line is activated by dialing the auto-answer unit. Trained obstetrical nurses at the EOPC monitor the data, make therapeutic suggestions according to protocols, and if threatening patterns occur, alert a perinatologist at the center or the referring physi-

Table 1. — Characteristics of Participating Hospitals

| Hospitals | Distance from tertiary center (miles) | Mean # deliveries per year | Total remote transmissions | % Deliveries in RFM program |
|---|---|---|---|---|
| A | 54 | 165 | 201 | 81 |
| B | 26 | 320 | 26 | 5 |
| C | 55 | 20 | 37 | 92 |
| D | 90 | 70 | 9 | 10 |
| E | 84 | 60 | 28 | 35 |
| F | 87 | 87 | 33 | 18.9 |
| G | 41 | 128 | 62 | 24.2 |

RFM = remote fetal monitoring

cian. These same nurses are cross-utilized to observe monitoring displays in our own inpatient labor and delivery unit.

Interpretation of abnormal patterns was made according to previously established criteria.[2] According to the clinical characteristics of each case, the following recommendations were made: continued observation, implementation of internal monitoring, immediate delivery, medical treatment (ie, oxygen therapy, positional changes), or patient transport. Follow-up information on maternal and neonatal outcome was obtained from each participating hospital. An ongoing continuing inservice training program was provided to participating hospitals by two highly trained obstetrical nurses.

## Results

Three hundred ninety-six remote fetal monitoring transmissions were received in the tertiary center from seven participating hospitals (Table 1). Participating hospitals joined the project at different times (mean 1.9 years, median 1.5 years). Total transmission time was 2,232 hours (mean time for transmission 338 minutes, SD 320 minutes). The types of fetal heart rate patterns diagnosed were: variable decelerations 58.6%, late decelerations 14.8%, early uniform decelerations 3.5%, prolonged decelerations 1.2%, decreased beat-to-beat variability 5.8%, bradycardia-tachycardia 3.1% each, and abnormal contraction patterns 3.75%. A saltatory pattern was found in one patient.

Recommendations for therapeutic intervention were made 302 times. The most common were: turn patient to left side, 109 times; give oxygen to the mother, 67 times; and immediate consultation with attending physician or perinatologist, 24 times. The overall cesarian section rate in the group monitored by telemetry was 11.1%. Seven patients (1.7%) had cesarian sections for fetal distress (Table 2).

Five-minute apgar scores were more than 7 in 93% of the patients. Postmaturity was the most frequent high risk factor identified (68 cases, or 16%). Premature rupture of membranes had the highest incidence of late decelerations (9 out of 17, or 52.9%). Oxytocin administration (augmentation or induction) was associated with 128 (32.3%) of the total 396 intrapartum tracings. Evidence of hyperstimulation was detected in 15 (11.7%) of these cases.

The incidence of prematurity (>34 and <37 weeks) was 8% (32 cases). Our analysis is based mostly on gestational age by dates and not on Dudowitz or sonographic criteria; the data on prematurity are, therefore, likely to be inaccurate. Late decelerations were diagnosed in 4 (12.5%) of the tracings of preterm labor. There were 8 patient transports as follows: premature labor 5; preeclampsia 1; cephalopelvic disproportion (CPD) 1; failure to progress 1. During the study period, there were 37 neonatal transports from the participating hospitals. Seven of the 37 infants were born to mothers enrolled in the Remote Fetal Monitoring Program (RFM). An abnormal fetal heart rate pattern was detected in 3 of these 7 mothers. The resulting neonatal condition in those three infants were: meconium aspiration, 1 case; respiratory distress syndrome (RDS), 1 case; and mild hypoxic ischemic encephalopathy, 1 case.

Table 2. — Cesarian Sections for Fetal Distress (N = 7)

| Maternal data | FHR | Apgar/outcome* |
|---|---|---|
| 42 weeks | Hypertonic contractions | 8/10 |
| | Late decel | NP |
| 39 weeks | Late decel | 8/9 |
| Prolonged labor | Bradycardia | NP |
| 41 weeks | Severe variables | 3/6 |
| BP | Late decel | Bag mask resusc |
| 40 weeks | Tachycardia | 4/6 |
| Induction | Late decel | Intubation resusc |
| | | RDS |
| 35 weeks | Late variable | Twin A 1/6 |
| Twin pregnancy | Hypertonic contractions | NP |
| | | Twin B 8/9 |
| | | NP |
| 38 weeks | Persistent late uniform | 2/6 |
| | | SGA |
| | | NP |
| 41 weeks | Late decel | 7/9 |
| | | NP |

*NP = No problems in newborn period

## Discussion

New advances in medicine are often imposed on smaller institutions without the benefit of consultation and support systems from more experienced institutions. The immense popularity of fetal monitoring, as well as its medical-legal implications, has prompted us to develop a consultation mechanism for small obstetrical units. Our system adapted the transtelephonic electrocardiography technique commonly used to monitor adult cardiac patients.[3] The use of a Xerox telecopier and the transtelephonic transmission of fetal monitor tracings have been described.[4,5] Those telemetry systems, although regional in scope, did not offer "on-line" surveillance. The immediate recognition of threatening FHR patterns by experienced health

professionals is a clear advantage of our system.

The most frequent problem associated with an abnormal intrapartum FHR patterns uncovered by this study was postdatism. We are therefore directing our efforts to further educate health professionals about this obstetrical problem.

We have also found uterine hyperstimulation secondary to improper use of oxytocin. It is expected that the development and use of low-dose oxytocin protocols will solve this problem.

Another problem identified by our survey was the failure to implement internal monitoring when external monitoring displayed excessive artifacts. The inordinately high rate of prematurity found in the study prompted us to teach the Dubowitz scoring system in our outreach educational program. The hospitals participating in the program varied in their requirements. Larger units utilized the service to increase the sophistication of nurses and physicians in interpreting difficult tracings. Smaller units with fewer deliveries and limited personnel often relied on our program as a means to monitor their patients.

We believe that the major advantage of this program lies in its great educational potential and in maintaining lines of communication among all members of the obstetrical care system. It is the authors' opinion that the Remote Fetal Monitoring Program can decrease neonatal mortality and morbidity rates in participating geographic regions, although the present study did not endeavor to prove this contention.

## Summary and Conclusions

The remote fetal monitoring (RFM) program is a consultation and support system designed to foster interaction between health professionals in remote rural areas and those in tertiary centers. The benefits of this program are realized in the instantaneous recognition by trained personnel of nonreassuring fetal monitoring patterns and in the immediate availability of perinatal consultation.

The RFM program has the following advantages:

1. Permits rural obstetrical units to have access to continuous fetal monitoring despite limited manpower.
2. Allows the discovery of specific areas where educational efforts should be directed.
3. Helps in identifying those obstetric patients in need of care in a tertiary center.
4. Improves the quality of care in small obstetrical units.

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# Psychiatric Treatment of Erectile Dysfunction

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**In the last two decades, significant advances have been made in the diagnosis and treatment of erectile dysfunction (impotence). The publication of *Human Sexual Inadequacy* in 1970 ushered in a paradigm shift. The concepts that sexual behavior is a learned skill and that the sex therapist functions as a kind of athletic coach changed the field. In the last decade, however, this approach has been enriched by concepts and techniques from marital and family systems theory.**

Erectile dysfunction is defined in the current *Diagnostic and Statistical Manual* (DSM III) of the American Psychiatric Association as recurrent or persistent inhibition of sexual excitement that is manifested by partial or complete failure to attain or maintain erection until sexual orgasm, despite the fact that the individual engages in sexual activity that is adequate in focus, intensity, and duration. This latter qualification is important because a man needs more stimulation to attain and maintain a firm erection as he ages. If he does not appreciate this, he may mislabel himself as "impotent."

This diagnosis excludes men with an exclusively organic etiology, although, in reality, many impotent men have both organic and psychiatric components to their dysfunction. It also excludes those with some other primary psychiatric diagnosis, such as depression, anxiety, or drug dependence.

The current (1980) *Diagnostic and Statistical Manual* (DSM III) includes five axes along which each patient is diagnosed. These are:

| | |
|---|---|
| Axis I. | Clinical psychiatric syndrome |
| Axis II. | Personality disorder or specific personality traits |
| Axis III. | Physical conditions |
| Axis IV. | Severity of psychosocial stressors |
| Axis V. | Highest level of functioning during past year |

It is easy to see how useful these five axes are in the case of erective dysfunction. When a man has a physical illness that is responsible for some of his problems — hormonal, vascular, or neurological — or is taking drugs which can cause impotence, these are listed on Axis III. Heavy use of tobacco is one of these conditions, although rarely recognized as such — a truth which should certainly add power to any antismoking campaign.

With the introduction of this new system of diagnostic classification in 1980, it became possible to separate out a whole new group of sexual disorders, the disorders of sexual desire, which previously had been lumped with "impotence." This has been a major advance, since these conditions are

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generally more complex and involve more psychological and marital factors than most cases of erectile dysfunction. It is also the most frequent condition for which men come to sex therapists and now constitutes about 40% of most case loads.

## Historical Perspective on Sexual Dysfunctions

Degeneracy was the most generally accepted etiology for sexual dysfunctions at the turn of the century. Both excessive masturbation and excessive intercourse were believed to lead to irreparable harm. Graham crackers were introduced as bland foods to help people calm their sexual urges and thereby avoid slipping into moral degeneracy. Right up until the 1930s, combinations of the chastity belt and the strait-jacket were sold to curb "the deadly vice of onanism."

Freudian theory postulated that sexual dysfunction resulted from a failure to accomplish childhood developmental tasks, a view which held sway long after it was challenged by research. In 1958, Joseph Wolpe conceptualized sexual dysfunctions as conditioned anxiety responses to sexual situations, and prescribed a treatment for impotence which was markedly similar to that described by Sir John Hunter in the eighteenth century: the patient was instructed to lie naked with his partner, but forbidden to proceed to intercourse.

In 1956, Seemans described the "start-stop technique" for the treatment of premature ejaculation, a method which, if followed, results in a virtually 100% cure rate. In 1962, Albert Ellis delineated an approach that incorporated cognitive therapy, education, and skill-training. However, belief systems are not necessarily changed by facts.

**For about 20% of the men we see, the "impotence" is situation-specific.**

It was only in 1970, with the publication of *Human Sexual Inadequacy* by Masters and Johnson that a paradigm shift occurred. The authors' position that sex is a learned skill, and their conceptualization of the sex therapist as a kind of athletic coach changed the field.

The later seventies and the early eighties brought into sex therapy an integration of family and systems theories. In part, this was brought about by the fact that, as sex therapy techniques became better known in the medical community, sex therapists began to see more complicated cases: those for whom the simpler behavioral techniques had already been tried.

A number of problems continue to plague the field. For research purposes, the DSM III classification is inadequate. Consequently, each research center has tended to use its own system of classification, and these systems have not necessarily been compatible. Second, many groups have put together treatment packages, which makes it difficult to ascertain the impact of any single therapeutic variable.

## Diagnosis

First, a thorough physical work-up must be done. This is beyond the scope of this paper, but includes hormonal, neurological, and vascular studies. Recent advances have demonstrated that a number of men who previously would have been diagnosed as suffering from psychological impotence do indeed have a major organic component to their dysfunction. Davis et al, for example, found co-existing medical conditions of importance in 90% of the 93 men over 50 years of age whom they treated. However, the existence of significant organic factors does not rule out the value of sex therapy. The problems of a man with diabetic neuropathy, for example, may very well be compounded by secondary psychological factors.

The psychiatric work-up begins with a very detailed history, with considerable emphasis on specific sexual knowledge, activities, attitudes, and reactions. The couple is seen together, for there may be major marital issues involved in the problem. The woman may be ambivalent about intercourse and her husband's impotence may be helping her to avoid it, while reassuring her that he is not involved with other women. He may also be more solicitous because of his guilt. These are all advantages she may wish to retain. "You owe it to me because I've stayed with you despite your impotence" may be a source of power she is reluctant to renounce.

It is also important to see each member of the couple individually. Neglecting to do this may be to invite one spouse either to lie or to "drop a bomb" on the other. Privately, they can be asked about premarital and extramarital sexual activity, sex fantasies and preferences, and the possibility of divorce. It is important to know whether each

actually loves the other and whether each finds the other sexually attractive and skilled in bed.

For about 20% of the men we see, the "impotence" is situation-specific: they are impotent with their wives, but not with their mistresses. Obviously, this is not a case for traditional sex therapy of a behavioral variety.

A not-uncommon scenario is that of the recently divorced, middle-aged man who goes to a party where he is picked up by a divorcee. (At age 40, there are twice as many single women as men; at age 50, four times as many.) When they finally go to bed, his stomach is full, he has drunk too much, smoked too much, and is more than a little uneasy because of her assertiveness. He finds himself unable to perform. If he does not accept this as situation-specific, but instead mislabels himself "impotent," the next time he may badger himself with a constant internal dialogue of self-harassment, such as, "Wouldn't it be awful if. . ." and "What if. . .," or may torture himself with judgments of his performance and fantasies of how his partner may judge it — both destined to interfere with his abandoning himself to the enjoyment of the moment.

It is also not uncommon to find a widower who has become attached to his wife's best friend, or to a single neighbor who helps him through the last stages of his wife's illness and her funeral. When asked privately, he can usually tell you he is not through mourning his wife's death, does not really "love" his new friend, but does not feel free not to go to bed with her because "she is a fine woman, and I don't want to hurt her feelings."

## Treatment

Although a number of sex clinics offer therapeutic packages, there is no one right way to treat these people. The best way depends on the patient, his partner, their situation, and the personality and expertise of the therapist. In general, however, all treatment plans involve some mixture of the following:

1. **Education**
   Many patients lack basic sexual information. Some men and their spouses are unaware that as a man ages he will need more physical stimulation, nor do they know how to supply it.

2. **Communications Training**
   Many individuals do not feel free to share with their partner what they find stimulating, what would be most exciting to them, or what they do not want.

3. **Behavioral Techniques**

   *(a) Pleasuring and decreasing performance anxiety*

   The couple are instructed to spend some time giving one another a leisurely sensuous body massage, but forbidden to have intercourse. This removes the pressure of needing to perform and gives them the opportunity to explore giving and receiving pleasure. It also tends to interrupt obsessive internal dialogue, self-harassment, and "spectatoring," the standing off mentally and grading one's performance which commonly is a feature of this condition.

   At a later time, they are instructed to incorporate genital stimulation. At the very least, this technique interrupts obsessive ruminations, relieves the pressure to perform, gives permission to give and receive pleasure, and distracts. Some men find the sensuous stimulation in a non-demanding atmosphere so stimulating that they "disobey" instructions and spontaneously proceed to intercourse.

   *(b) Teasing*

   The penis and perineal area are stimulated until an erection results, then the stimulation is temporarily stopped, to be resumed again as the erection subsides. How this is done depends on the sexual repertoire acceptable to the couple.

   *(c) Utilizing REM-stage erections*

   When a man has shown a lifelong lack of responsiveness, it may be possible to take advantage of the erections that occur during REM sleep. His partner is instructed to begin fondling his penis when it becomes erect, so that he is awakened aroused.

4. **Cognitive Therapy**
   Some men maintain their erective dysfunction through their thinking processes — what they say to themselves in their heads, and through their underlying belief systems. These processes can be addressed directly through cognitive therapy.

These techniqes are usually effective, if followed. However, the couple may "forget" to practice them, be "too busy," or may do them incorrectly, despite

careful coaching. A sensuous massage should not be performed in the manner of a "rub-down" at the YMCA. When these "resistances" arise, it is an indication that the therapist should look at more psychodynamic and systemic issues.

## Individual Factors

Men with clear psychiatric symptoms such as depression, anxiety, or drug dependence should be treated for these conditions before sex therapy is initiated. More subtle, however, are a man's attitudes and beliefs. Some men, for example, do not believe the marriage is really consummated until they ejaculate in the vagina.

In considering individual psychodynamic factors that may need to be addressed, it is useful to give special attention to the following four areas:

1. Internal freedom of fantasy and behavior during intercourse
2. Ability to conceptualize and to describe oneself and others in depth, a deficit which is found in lower-level personality disorders.
3. Quality of superego formation, as manifested in ability to idealize, fall in love, evidence concern for and commitment to another, as well as in unconscious prohibitions
4. Discrepancy between conscious and unconscious sexual orientation

## Marital/Systemic Factors

The past ten years have seen an explosion in our knowledge of family systems and in therapeutic techniques that work at this level. Current family structure, repetitive interactional patterns, response to external stressors and life-cycle changes, individual goals and aspirations for the marriage, the unconscious projection of parental images on the spouse, as well as the transgenerational transmission of "hot topics" and induction of roles — techniques to deal with these are now all part of the repertoire of the family therapist. The major issues are usually those of trust, intimacy, anger/hurt, and power. Unresolved problems in these basic areas may be giving one of the partners an advantage in the relationship, and erectile dysfunction can be one symptom of this. The chances of therapeutic success in treating the dysfunction are limited if the underlying conflict is not resolved.

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*James Ross Allen, MD, is a clinical professor of psychiatry at the University of Oklahoma Tulsa Medical College. A 1961 graduate of the University of Toronto, he is certified by the American Board of Psychiatry and Neurology and the American Psychiatric Association Committee on Certification in Administrative Psychiatry.*

## Coming in February . . .

The second part of the paper "Thyrotoxic Hypokalemic Periodic Paralysis," begun in this issue, will appear. Being considered for publication are a discussion of glycosylation in the assessment of diabetic control and a report of a case of Adie's syndrome.

## News from the Oklahoma State Department of Health

# Wellness Check for Teens

During the past year, the Oklahoma State Department of Health has been involved in a new health education program for high school students. The voluntary program, called Wellness Check, is based on a computer-scored questionnaire which helps determine a student's health status. The Wellness Check program covers a wide variety of health topics ranging from nutrition and exercise to safety and seat belt use.

Students answer the questionnaire on a computer card which is optically scanned by a microcomputer. Within seconds, the answers are tabulated and an individualized printout is given to the student. Based on the student's answers, a wellness score from 0 to 100 points is determined, and the student is placed in one of four wellness categories — excellent, fair, risky, or hazardous. Most important, health risks within the student's life-style are identified, and suggestions are made on actions the student may take to live a healthier, longer life. These positive health actions are described in detail in an accompanying booklet called "The Way to Wellness." To answer concerns of the students and to refer students to community health resources, counseling is provided by health department staff at the time of the computer analysis.

So far, over 1,100 high school students across Oklahoma have participated in the Wellness Check program. Summary data show that of these 1,100 students, 90% do not always wear seat belts. An alarming 40% indicated that they drive or ride in a vehicle under the influence of alcohol, and 39% admitted to drinking at least some alcohol. Other areas of concern include poor dietary habits (66%), lack of exercise (54%), and high stress levels (21%).

As additional data are collected, health department officials hope to identify specific areas of risk for which effective health education intervention models can be developed. Currently, various avenues are being explored which will allow the expansion of this worthwhile and important program. Physicians who would like more information about this program may contact Neil E. Hann, MPH, Health Education and Information Service at (405) 271-5601.

| DISEASE | October 1986 | TOTAL TO DATE | | |
|---|---|---|---|---|
| | | This Year | Last Year | 5 Yr. Avg. |
| AMEBIASIS | 0 | 7 | 12 | 13 |
| CAMPYLOBACTER INFECTIONS | 23 | 234 | 263 | — |
| ENCEPHALITIS, INFECTIOUS | 1 | 20 | 26 | 28 |
| GIARDIA INFECTIONS | 40 | 206 | 287 | — |
| GONORRHEA (Use ODH Form 228) | 1257 | 10753 | 11076 | 12543 |
| HAEMOPHILUS INFLUENZAE INVASIVE DISEASE | 27 | 196 | 208 | — |
| HEPATITIS A | 30 | 300 | 404 | 480 |
| HEPATITIS B | 21 | 172 | 192 | 228 |
| HEPATITIS, NON-A NON-B | 4 | 50 | 62 | — |
| HEPATITIS UNSPECIFIED | 8 | 42 | 72 | 140 |
| MEASLES (RUBEOLA) | 0 | 39 | 1 | 9 |
| MENINGITIS, ASEPTIC | 17 | 117 | 130 | 170 |
| MENINGITIS, BACTERIAL (non-meningococcal, non H. Influenzae) | 5 | 70 | 59 | 51 |
| MENINGOCOCCAL INFECTIONS | 6 | 27 | 26 | 29 |
| PERTUSSIS | 13 | 117 | 181 | 147 |
| RABIES (Animal) | 4 | 57 | 95 | 133 |
| ROCKY MOUNTAIN SPOTTED FEVER | 17 | 100 | 93 | 122 |
| RUBELLA | 0 | 0 | 1 | 1 |
| SALMONELLA INFECTIONS | 56 | 433 | 389 | 410 |
| SHIGELLA INFECTIONS | 21 | 188 | 249 | 297 |
| SYPHILIS (Use ODH Form 228) | 19 | 134 | 179 | 172 |
| TETANUS | 0 | 1 | 1 | 1 |
| TUBERCULOSIS | 26 | 217 | 210 | 238 |
| TULAREMIA | 2 | 11 | 17 | 25 |
| TYPHOID FEVER | 0 | 2 | 2 | 3 |

| Diseases of Low Frequency | Total to Date This Year |
|---|---|
| ACQUIRED IMMUNE DEFICIENCY SYNDROME | 31 |
| BRUCELLOSIS | 0 |
| LEGIONNAIRES DISEASE | 17 |
| MALARIA | 10 |
| REYE SYNDROME | 5 |
| TOXIC SHOCK SYNDROME | 25 |

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# State lawmakers investigate Medicaid claims problems

Left: Robert Fulton, director, Oklahoma Department of Human Services (DHS). Right: Mike Fogarty, DHS director, medical services.

Left (pointing): Senator Stratton Taylor, D-Claremore. Center: Senator Ged Wright, R-Tulsa. Far right: Senator Bill Dawson, D-Seminole.

Committees from both houses of the Oklahoma Legislature held special meetings this fall to investigate complaints from Oklahoma physicians and other providers about significant problems and delays in processing Medicaid claims submitted to the Oklahoma Department of Human Services (DHS).

Senator Stratton Taylor, D-Claremore, chaired the Senate committee and Representative Don Mentzer, D-Sallisaw, lead the House group.

Dozens of physicians and office managers testified about claims returned, rejected, suspended, and delayed. Many had waited months for payment.

On April 1, 1986, SDC Corporation began processing claims for the DHS. SDC representatives blamed initial shakedown of the system and unfamiliarity with new forms and coding requirements for the problems.

During their 1987 session, Oklahoma legislators will review SDC's progress in alleviating the problems.

# Trustees approve fourteen OSMA Life Memberships in November

At its November 16 meeting, the Oklahoma State Medical Association Board of Trustees approved Life Memberships for the following members:

From Oklahoma City, Jones B. Ballina, MD; James P. Bell, MD; Glenn P. Dewberry, Sr., MD; J. B. Eskridge III, MD; Lillian M. Hoke, MD; Pamela R. Parrish, MD; and Lindbergh J. Rahhal, MD.

From Tulsa, William R. Coutant, MD; Harlan Thomas, MD; and Clayton E. Woodard, MD.

Also approved were James M. Fite, MD, Muskogee; Woodrow W. Massad, MD, Ponca City; Ethel M. Walker, MD, Harrah; and Avery B. Wight, MD, Enid.

To be eligible for a Life Membership, an OSMA member must meet one or more of the following qualifications: (1) Be retired from the active practice of medicine due to ill health or age; (2) Be engaged in the active practice of medicine for fifty years or more; (3) Be seventy years of age or older.

*Physician discipline discussed*

# Select Committee meets again, hears testimony from Dr Long

Oklahoma's new Select Committee on Insurance Rates and Tort Claims met again on Thursday, December 4, in Oklahoma City. Among those addressing the committee was Larry Long, MD, speaker of the OSMA House of Delegates.

The committee, consisting of ten legislators and eight lay members, is charged with studying, preparing recommendations, and drafting legislation on the problem of increasing liability premiums and court awards.

Following is the text of Dr Long's address.

### Physician Discipline

It has been said that the medical profession shrouds itself in a "conspiracy of silence," protecting each other rather than the public from dated, duped, or dishonest practitioners.

If that ever was true, it is not the case any longer.

Today medicine is the most scrutinized of professions.

The conduct and competence of physicians are examined and criticized by their peers, state licensing boards, hospital credentialing committees, hospital morbidity and mortality studies, hospital tissue committees, federally mandated peer review organizations, private health insurance companies, government health insurance programs, malpractice insurance carriers, county medical societies, state medical associations, national specialty groups, and the American Medical Association.

To paraphrase the late, great Joe Louis, the substandard physician may be able to run for a short while but he no longer can hide. There are, simply and quite correctly, too many eyes watching everything a physician does. No other profession, in my opinion, is subject to such rigorous and intense review by so many sources — colleagues, federal and state laws and regulators, state boards and bureaus, hospitals, the insurance industry, and, most of all, the public.

Fact: In statistics just released, the Federation of State Medical Boards of the United States reports that 60% more physician licenses were revoked in 1985 than ever before. Last year 406 physicians had their licenses revoked, 491 licenses were suspended, and 976 doctors received penalties ranging from reprimands to practice restrictions.

Fact: The Oklahoma State Board of Medical Examiners, one of the nation's most respected, is actively investigating, at any given time, more than 100 cases.

Fact: In the first three quarters of 1986, the Oklahoma State Board of Medical Examiners has taken 38 actions against substandard practitioners including three license revocations, two suspensions, 11 probations, three formal reprimands, and 19 other actions ranging from restricted prescription writing to mandated treatment for chemical abuse.

Fact: The Oklahoma State Medical Association Physician Recovery Program seeks to identify and secure treatment for physicians with alcohol or chemical dependency problems. Currently the OSMA Physician Recovery Committee is supervising the treatment and recovery of over 100 physicians.

Fact: The Oklahoma Foundation for Peer Review, which holds the Health Care Financing Administration (HCFA) contract to assure the quality of care offered to Medicare patients in Oklahoma, reviews thousands of medical cases every year. In the past 18 months, 41 cases have been reviewed for "substantial"

(continued)

## Medicine Day is coming February 18

Medicine Day at the State Capitol, the jointly sponsored project of the OSMA and the OSMAA, will be held on Wednesday, February 18, 1987. Medicine Day will begin at 9:00 am with registration and orientation taking place in the Oklahoma House of Representatives' Chamber. The day will allow participants the opportunity to meet with their respective representatives and senators as well as to help promote the more positive aspects of medicine. Various specialty societies will be setting up booths in the Capitol Rotunda for this special day. Every interested physician, physician spouse, office staff member, nurse, and community leader is urged to attend this all-important event.

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*L. O. Laughlin, MD, to head OSIM*

# State internists elect new president from Oklahoma City

An Oklahoma City physician is the new president of the Oklahoma Society of Internal Medicine (OSIM).

L. O. Laughlin, MD, an internal medicine specialist in Oklahoma City since 1966, was elected during the OSIM Annual Meeting in October at Shangri-La Resort in Afton. His term expires in October 1988. Dr Laughlin succeeds Dr John Kalbfleisch of Tulsa.

Board eligible in internal medicine and nephrology, Dr Laughlin was graduated from the University of Oklahoma School of Medicine in 1956. He was the first practicing nephrologist in Oklahoma City and developed the first transplant program in a private hospital in the state. He also established the first satellite dialysis unit in Oklahoma. He is currently the medical director of the Presbyterian Hospital dialysis unit.

J. Kin Pirtle, MD, Norman, was named vice-president and president-elect, and Michael Strange, MD, Stillwater, Eldon Gibson, MD, Shawnee, and James Schmidt, MD, Oklahoma City, were named to the governing council.

The Oklahoma Society of Internal Medicine has approximately 260 physician members in Oklahoma who specialize in the practice of internal medicine. The purpose of the society is to maintain the highest standards of internal medicine for the benefit of the patient by sponsoring continuing medical education programs and providing internists with a voice on vital scientific and socioeconomic issues.

OSIM is a component of the American Society of Internal Medicine (ASIM), a federation of 51 societies representing over 20,000 internists nationwide.

## Select committee (continued)

deviation from quality standards and appropriate remedial action has been undertaken.

Fact: The Underwriting Committee of PLICO, Oklahoma's physician-owned professional liability insurance company, routinely reviews the practice patterns of any physician with a minimum of three claims.

Fact: In the last year alone, the PLICO Underwriting Committee has conducted intensified review of 125 physicians resulting in loss or decrease of insurance for 13 physicians.

The facts, at least in Oklahoma, are clear. Medicine, more than any other profession, both welcomes and encourages the scrutiny necessary to ensure high quality medical care, and works actively to protect the public from substandard practitioners and thus maintain the dignity, trust, and honor of the medical profession.

*Respectfully submitted,*
*Larry L. Long, MD*
*Member*
*Select Committee on Insurance Rates and Tort Claims*

*Many issues unresolved*

# JAMA editor warns of hazards in mandatory drug testing

Urine screening for drug use can be done very accurately, but serious technical, moral, economic, and civil liberties issues must be addressed before random, mandatory drug testing makes sense, the editor of the *Journal of the American Medical Association (JAMA)* warns.

If society feels drug abuse is so serious a problem as to justify the loss of individual freedoms, George D. Lundberg, MD, writes in an editorial, mandatory, random urine drug screening can be accomplished. But such a decision, he says, should be made by "an informed electorate, probably through the state-by-state referenda. . . . The issue is that important."

Drug use "continues to be a major phenomenon of our time," Lundberg says. He notes that despite all anti-abuse efforts to date, "psychoactive drugs continue to be widely available at relatively low cost and are widely used" and says the age of "designer drugs" can only compound the problem.

While urine drug screening technology has improved greatly in recent years, its routine application may not have, Lundberg says. Survey data from the Centers for Disease Control revealed "woefully poor performance by many laboratories on blind testing," while data from the College of American Pathologists indicate false-positives on confirmatory tests "continue to appear in distressingly large numbers," he notes.

Even the best screen can't be guaranteed all the time, Lundberg says, and a fail-safe system of guarding against tampering with samples is

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## Mandatory drug testing (continued)

unlikely. What's more, he adds, drug testing technology is expensive. The US military's drug screening program costs an estimated $90 to $100 per sample, he notes; applying a similar approach to the American workforce — at a single sample per worker per year — would cost society $8 to $10 billion a year.

While pleased by the symbolic importance of the new billion-dollar federal "war against drugs," Lundberg sees little chance it will have a major impact on any large group in society "other than politicians and bureaucrats." In addition, he argues, hypocrisy in drug use "continues unabated . . . the drugs that most often kill are legal, and far less toxic drugs are illegal. . . . It is also clear that the most commonly lethal drugs (ethyl alcohol and tobacco) continue not to be part of urine drug screening in most settings."

Compounding the scientific and economic issues, Lundberg writes, is the fact that the main purpose of such testing, in the absence of clinical indications, is legal — and a host of legal and civil liberties issues remains unaddressed. Just one such issue yet to be worked out by the courts, he says, is "whether any individual who is apparently functioning normally with no demonstrated impairment can be subjected to a form of intimate body search."

Lundberg says he has no doubt that "the realization that observation in the form of urine drug screening may occur will prevent many healthy people from using illegal drugs, saying, "we should keep it rolling."

"However," he writes, "the adverse effects of excessive zeal on individuals, organizations, and society can be enormous. We should not perform actions that are as likely to cause harm as good."

In 1972, while involved in drug abuse toxicology at the University of Southern California School of Medicine, Lundberg recalls writing a letter to the *New England Journal of Medicine* calling urine drug testing "Chemical McCarthyism," citing the tangle of issues involved. While technology may have improved since then, he concludes, "the more things change, the more they remain the same."


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**Free pizza** helped to draw more than 80 medical students to their December 2 meeting in Oklahoma City. Of the 26 pizzas provided by the OSMA, not a single slice was left. Highlight of the meeting, however, was a showing of the OSMA film *Preserving Tradition, Embracing Change.*

*Physicians feeling pressure*

# Survey examines changes in doctor/patient relationship

Almost half of American physicians believe they are unduly pressured to release Medicare patients early from the hospital, according to a nationwide survey commissioned by the American Medical Association (AMA) and reported in *American Medical News.*

Of the 1,000 physicians surveyed by the AMA in June, 48% said they felt "unduly pressured" to discharge Medicare patients early, while 28% felt no pressure. The remainder were unsure or did not answer.

Physicians serving patients at opposite ends of the population spectrum more often reported feeling pressed to release their patients early: 58% of doctors practicing in counties with less than 25,000 people and 59% of the physicians in counties of five million or more people.

"Reimbursement of hospitals by Medicare is currently based on the diagnosis related group (DRG) into which a patient's illness fits. This plan generally provides a particular level of payment for all patients within one DRG, regardless of differences in length of hospital stay," said James H. Sammons, MD, AMA executive vice president. "Thus, there may be an economic incentive for the hospital to limit the length of stay of any Medicare patient, and physicians may feel some pressure to discharge Medicare patients as soon as possible."

Fifty-six percent of the doctors said they felt their control over patient treatment decisions in the hospital had decreased in the last several years, while 6% said it had increased, and 38% said their control over decisions was unchanged. The feeling of less control was especially pronounced among obstetrician/gynecologists (66%), but also common among surgeons, psychiatrists, and general practitioners. Of doctors practicing in counties of less than 25,000 population, 67% reported a decrease in control.

The doctors also were asked whether patients had changed over the last three years. Seventy-six percent said patients are now "more knowledgeable about factors contributing to health" and "more concerned about costs of treatment." Only 3% of those surveyed said patients were less knowledgeable, and 21% said patient knowledge was unchanged. Four percent said patients were less concerned about costs and 20% said cost concerns were unchanged.

Sixty-three percent of those surveyed believed patients are more demanding of their doctor than three years earlier; only 3% said patients had become less demanding. Thirty-four percent said patient demands remained the same.

Half the respondents said it was just as difficult to manage a patient clinically as it was three years ago. Forty-three percent said it was more difficult,

*Questions include Medicare funding*

# AMA polls public about life support systems, other issues

Nearly 75% of American adults favor withdrawing life support from hopelessly ill patients under certain conditions, overwhelming majorities favor three alternatives to financing health care for the elderly, and two-thirds believe the government should pay for health care for the homeless, according to an American Medical Association national survey reported in *American Medical News*.

Of the 1,510 respondents polled in May and June, 73% favored "withdrawing life support systems, including food and water, from hopelessly ill or irreversibly comatose patients if they or their family request it." Only 15% were opposed and 12% were unsure.

"Nearly three out of four favoring the withdrawal of life support in such dire circumstances is a reassuring endorsement by the American public of a decision made last spring by the AMA's Council on Ethical and Judicial Affairs," said James H. Sammons, MD, executive vice president of the AMA.

Sixty percent of the public believes payments for medical care for the elderly under the Medicare program should be based on the patient's income. Reflecting current Medicare policy, 35% of the respondents believe Medicare patients should be reimbursed for their health care "regardless of how rich or poor they are." Basing reimbursement on income was marginally more popular among the better educated and more affluent and was less favored by those over age 65.

If additional funding were needed to keep the Medicare system from going bankrupt, 70% said they would be willing to pay additional taxes to assure themselves of Medicare benefits when they reached age 65. However, 23% said no. Again, older respondents were not as willing to pay higher taxes to continue Medicare.

Almost eight out of ten respondents would be willing to make tax-deductible contributions to


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## Survey examines changes (continued)

and 7% thought it less difficult. One-fourth of the doctors said their patients were less satisfied with the treatment process, 27% said they were more satisfied, and 48% said patient satisfaction was unchanged. According to 23% of the physicians, patients are now more likely to follow prescribed treatment. Eighteen percent said patients were less likely to comply, and 59% believed compliance remained constant.

Among specialists, psychiatrists were most likely to view their patients as more cost-conscious. Anesthesiologists and obstetrician/gynecologists believed most strongly that patients are more demanding of their doctors, and the latter were most likely to say patients were more satisfied with treatment. Radiologists most often reported that patients are harder to manage clinically.

The doctors surveyed were selected randomly from the AMA's Masterfile of all active US physicians. The sample included appropriate percentages of both AMA members and non-members and doctors of different ages and sexes. The interviews were conducted by telephone by Tarrance, Hill, Newport and Ryan of Houston.

individual health retirement accounts to supplement the medical benefits available through Medicare. Twenty-three percent of those surveyed said they would not make such contributions. Despite the high acceptance level by the survey population, those over age 65 were less interested in this option.

The concept of "Health IRAs" was endorsed by the AMA House of Delegates at its 1986 annual meeting as part of a comprehensive proposal to ensure adequate financing of health care for the elderly. Other elements of the proposal included limits on out-of-pocket spending by the elderly based on income, and a small tax increase for some segments of the population still at work.

When reminded of media reports identifying many discharged mentally ill patients as "homeless street people," two-thirds of those surveyed said the government should pay the health care for the homeless. Although 15% were not sure who should pay for the care, 11% said private charities should fund it. Five percent said the mentally ill people should pay for their own health care. Two percent said hospitals should cover the costs. One percent said the physicians should pay.

This survey involved telephone interviews with 1,510 adults, using a random sample of all residential telephones in the United States. The interviews were conducted over a three-week period by Kane, Parsons & Associates of New York.

## In Memoriam

### 1985

| | |
|---|---|
| *William Hampton Garnier, MD* | *November 20* |
| *Jesse Ray Waltrip, MD* | *November 30* |
| *Charles F. Obermann, MD* | *December 30* |

### 1986

| | |
|---|---|
| *Alexander Poston, MD* | *January 3* |
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| *Edward L. Leonard, MD* | *February 14* |
| *William C. Tisdal, MD* | *February 24* |
| *Donovan Dillon Mosher, MD* | *April 4* |
| *Fred D. Switzer, MD* | *May 10* |
| *John D. Jennings, MD* | *May 12* |
| *Phillip Wade Jones, MD* | *May 18* |
| *Herbert L. Owen, MD* | *May 28* |
| *Marianne Elsbeth Kosbab, MD* | *June 13* |
| *William W. Rucks, Jr., MD* | *June 27* |
| *Ralph A. Smith, MD* | *July 27* |
| *Howard D. Tuttle, MD* | *August 3* |
| *Welborn W. Sanger, MD* | *September 19* |
| *William Carl Ewell, MD* | *September 20* |
| *Marcella Steel, MD* | *October 1* |
| *William Pat Fite, Jr., MD* | *October 30* |
| *Samuel Jackson McDaniel, MD* | *November 2* |
| *Iron Hawthorne Nelson, MD* | *November 12* |

## Deaths

### William Pat Fite, Jr., MD
### 1922 - 1986

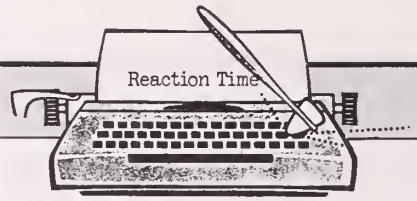
Tulsa plastic surgeon W. Pat Fite, Jr., died October 30, 1986. Dr Fite, a native of Muskogee, earned his medical degree at the University of Virginia School of Medicine in 1947. He was on active duty with the US Army for two years, attaining the rank of captain before his discharge in 1954. Dr Fite subsequently established practices in both Muskogee and Tulsa.

### Samuel Jackson McDaniel, MD
### 1917 - 1986

S. J. McDaniel, MD, a general practitioner in Oklahoma City, died November 2, 1986. Dr McDaniel was born in Springville, Miss, and graduated from the University of Arkansas School of Medicine in 1947. He practiced for several years in Waurika before his service in the US Army; he was discharged in 1955 as a captain and went on to establish a medical practice in Oklahoma City.

### Iron Hawthorne Nelson, MD
### 1899 - 1986

Retired pathologist Iron H. Nelson, MD, of Tulsa died November 12, 1986. A 1940 graduate of the University of Oklahoma School of Medicine, Dr Nelson was born in Randolph, Kan, and later served on active duty during World War I. He established his practice in Tulsa after graduating from OU and in 1981 was honored as Doctor of the Year by the Tulsa County Medical Society and TCMS Auxiliary.




# *Washington Post* publishes letter from Ed Calhoon, MD

Editor Ben Cason
Weekly Journal of Medicine
*Washington Post*
Washington, DC

Dear Editor Cason,

I read with interest your September 30, 1986, health section on Cancer in *The Washington Post*. The Congress has appropriated much money in the "War on Cancer." Many institutions of higher learning in America and even some overseas are funded for research and treatment of this dread disease. The Director of The National Cancer Institute (NCI) and its executive committee make far-reaching and important decisions that affect cancer patients. The National Cancer Advisory Board (NCAB) is appointed by the President of the United States. This board meets several times each year to assist, concur, and aid in funding of grants to various research centers. The National Cancer Act of 1971 was the last great legislative impetus. Fifteen years later Cancer has not been conquered. Many confusing statistics by many experts cause heated debate about Cancer cures.

As with all Government-funded efforts, a well entrenched bureaucracy exists at NCI. Cancers of the Solid Tumor Type such as Breast, Prostate, Pancreas, Lung, Stomach, Colon, and Brain still lead to poor long-term results. Cancer cures are not the rule. Too much BASIC SCIENCE RESEARCH and not enough HUMAN RESEARCH tends to leave the practicing Physicians (community of America) wondering if grants in these areas are adequate. Greater emphasis on cause of malignancy would seem to be in order.

A recent editorial in the *Medical World News* (widely read by practicing Physicians and Surgeons) suggests NCI's favorable press releases often coincide with fiscal year budget approval from Office of Management and Budget. Seventy billion a year cost, incalculable mental anguish, pain, suffering, and horrendous loss of life (near 500,000/year) leaves me wondering if a greater consortium of Medical and Surgical thought from practicing Physicians should aid the Director of NCI.

Sincerely,
*Ed Calhoon, MD*
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*AMA Delegate of Oklahoma Delegation*
*Member on Council of Legislation AMA*
*Beaver, Oklahoma 73932*

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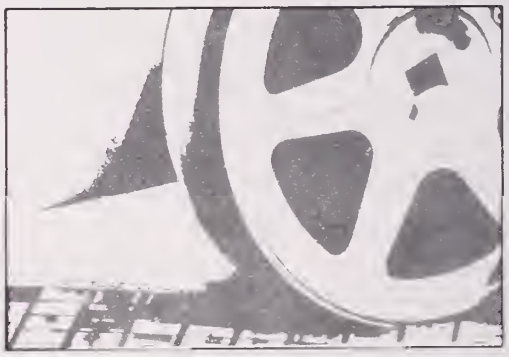

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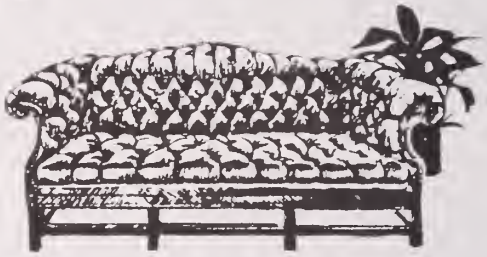

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# Challenge — Medicine Day Auxiliary Legislative Involvement

As the 41st Oklahoma Legislature is convened this month, many new members are taking their places in the House and Senate. The campaigns of last summer and fall behind them, these freshmen legislators now begin building their reputations as state policymakers. Along with incumbent and re-elected legislators, these freshmen are aware of the election trends and will be striving to achieve that elusive balance between personal goals and voter mandates.

On February 18, 1987, a unique opportunity exists for both auxilians and physicians, including their office staffs, to interact with the Legislature — MEDICINE DAY at the Capitol. A joint project of the Oklahoma State Medical Association and the OSMA Auxiliary, Medicine Day will provide a chance to promote the professional image of the physician through personal representation and educational displays. In addition, the cooperative efforts of the OSMA and Auxiliary will set a good example for county societies to follow in becoming involved with legislation.

The first stage of legislative involvement is a commitment of resources. For the physician this often involves personal and professional time in order to become educated in the process. In 1985, health care costs consumed over 10.7% of the gross national product and continued to grow at twice the overall inflation rate. With cost containment (at any price) the current focus of government relating to health care, the physician is finding the practice of medicine becoming a cookbook affair. Standards have been set for many procedures as to the inpatient/outpatient treatment mode and even recuperation days. Who will determine the assessment of this type of treatment in overall quality of care? As Mary Ann Deen, past president of the OSMAA stated, "Since government is now practicing medicine, it is time for medicine to begin practicing government."

The second stage of involvement is communication. By this, I mean the flow of information among the concerned parties — physicians, auxilians, legislators, lobbyists, patients, support staff, and others. All the facts about a particular issue need to be gathered and evaluated. If a piece of legislation with medical practice ramifications is drafted, it may then undergo significant changes from introduction to outcome. The tracking of legislation through its several stages is very important because of this. At each stage, input by the physician may be necessary. This input is vital to represent medicine's viewpoint. The more knowledgeable each physician is of this process, the easier it is to be effectively involved. At the state and national level, phone banks and mailgram alerts solicit physician and auxilian action.

The third stage of involvement is cooperation. Physicians are asked to be available to represent their views. Their patients can be sources as well as targets for information gathering and cooperative efforts. Auxilians can and do facilitate the spirit of cooperation through their networking efforts with other organizations both professional and social. The OSMAA, with its Legislative Action Committee, has undertaken a project to enhance their political knowledge and effectiveness. The end result will be a group that can assist their spouses in the increasingly political climate of medicine today.

I challenge you to begin the process of legislative involvement. Medicine Day at the Capitol is an excellent opportunity to do this. See you there!

*— Nadine Spring Nickeson*
*OSMAA Legislation Co-Chair*

■ **If you have not previously registered for** MEDICINE DAY at the State Capitol, time is running out! To register call Robert Baker or Mike Sulzycki at OSMA headquarters, 1-800-522-9452 or 405-843-9571. We look forward to seeing you on Wednesday, February 18, 1987.

■ **A report in the *Journal of the American*** *Medical Association (JAMA)* describes an improved system for reporting birth defects on birth certificates. Stephen D. Minton, MD, of the Utah Valley Regional Medical Center, and Robert E. Seegmiller, PhD, of Brigham Young University, say the new system "markedly improved the overall completeness of reporting while it identified inaccurate reporting of some malformations, incomplete reporting of multiple malformations, and the reporting of non-malformations as congenital malformations." Under the five-year-old system, responsibility for reporting defects is transferred from the mother's doctor to the newborn's doctor. A congenital malformation reporting sheet also is included in each newborn's file, and a centralized medical records person reviews the files and completes the certificate.

■ **Radiotherapy effectively controls Kaposi's** sarcoma and should be primary treatment for many cases of this rare skin cancer, says a study in *Archives of Dermatology.* Salah El-Akkad, MD, of the King Faisal Specialist Hospital and Research Centre, Riyadh, Saudi Arabia, and colleagues studied 13 Kaposi's sarcoma patients treated with radiotherapy from 1975 to 1984. Five were kidney transplant patients on immunosuppressive drugs; the rest developed the disease spontaneously. No case was AIDS-related. Of 11 patients followed for up to 63 months, all but one had complete response to radiotherapy, which was superior to chemotherapy in patients with spontaneous Kaposi's sarcoma. The study calls radiotherapy "a safe and effective form of treatment of Kaposi's sarcoma, with good relief of symptoms and minimal morbidity."

■ **Kidney transplant recipients and other** patients on immunosuppressive drugs are at increased risk of developing malignancies — often skin cancers — and should be closely monitored for this problem, notes a study in *Archives of Dermatology.* Aditya K. Gupta, MD, and colleagues at the University of Toronto, studied 523 white renal transplant patients — one of the largest studies of its kind in North America — and found 7.5% of them developed malignancies, 72% of these cutaneous in origin. Overall, the transplant recipients developed skin cancer 3.2 times as often as the general population and had a propensity for multiple skin cancers at an earlier age, especially in sun-exposed areas. "Immunosuppressed patients should be followed up with more vigilance than the general population to facilitate early diagnosis of skin cancer" and take extra precautions regarding sun exposure, the study concludes.

■ **Many surgeons now take a more conserva**tive approach to management of breast cancer, opting for lumpectomy and radiation therapy over modified radical mastectomy. But a report in *Archives of Surgery* suggests the more conservative approach also is more expensive. Eric Munoz, MD, MBA, of Long Island Jewish Medical Center, New Hyde Park, NY, and colleagues compared total treatment charges (hospital and physician) for 49 patients who underwent lumpectomies and 30 who had mastectomies during 1983 and 1984. Mean total charges per patient for lumpectomy were $14,176, compared with $10,345 for mastectomy. Lumpectomy patients had lower hospital inpatient fees but significantly higher mean total physicians fees. Radiotherapy charges also boosted the mean total charges for lumpectomy, the authors report.

■ **A study in *Archives of Ophthalmology* says** the number of intraocular lens implants performed annually in the US may be leveling off after years of rapid growth. Walter J. Stark, MD, of Johns Hopkins University, Baltimore, and colleagues note the number of implants performed has continued to rise in recent years but the rate of increase has slowed since February 1984. The number of implants performed between February 1985 and February 1986 topped 900,000 and should level off at just under one million a year if current trends continue, the study says. "The annual number of cataract operations appears to be adjusting to a new equilibrium rather than continuing the rapid increase of the first half of the decade," the study concludes.

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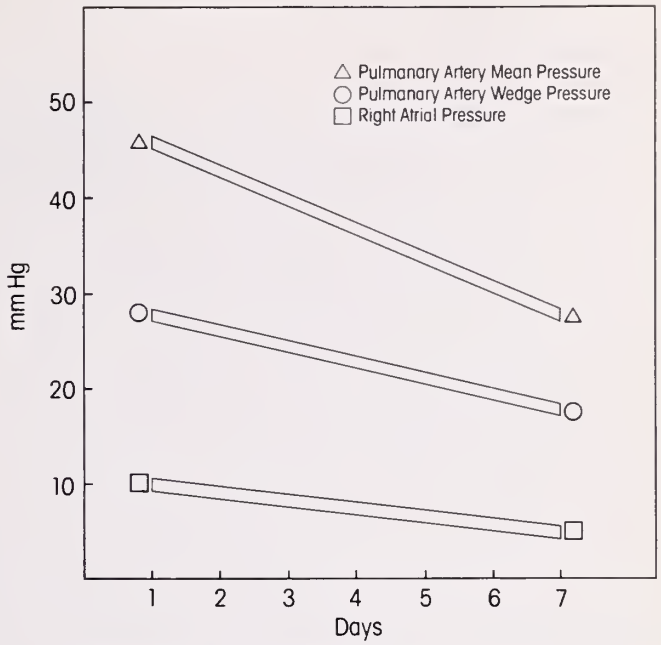


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**Before prescribing, please consult complete product information, a summary of which follows:**

**WARNING: Bumex (bumetanide/Roche) is a potent diuretic which, if given in excessive amounts, can lead to a profound diuresis with water and electrolyte depletion. Therefore, careful medical supervision is required, and dose and dosage schedule have to be adjusted to the individual patient's needs. (See under DOSAGE AND ADMINISTRATION in complete product information.)**

**INDICATIONS AND USAGE:** Edema associated with congestive heart failure, hepatic and renal disease, including the nephrotic syndrome.
Almost equal diuretic response occurs after oral and parenteral administration of Bumex. If impaired gastrointestinal absorption is suspected or oral administration is not practical, Bumex should be given by the intramuscular or intravenous route.
Successful treatment with Bumex following instances of allergic reactions to furosemide suggests a lack of cross-sensitivity.
**CONTRAINDICATIONS:** Anuria. Hypersensitivity and in patients in hepatic coma or in states of severe electrolyte depletion. Although Bumex can be used to induce diuresis in renal insufficiency, any marked increase in blood urea nitrogen or creatinine, or the development of oliguria during therapy of patients with progressive renal disease, is an indication for discontinuation of treatment.
**WARNINGS:** Dose should be adjusted to patient's needs. Excessive doses or too frequent administration can lead to profound water loss, electrolyte depletion, dehydration, reduction in blood volume and circulatory collapse with the possibility of vascular thrombosis and embolism, particularly in elderly patients.
Prevention of hypokalemia requires particular attention in patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis and ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy, certain diarrheal states, or other states where hypokalemia is thought to represent particular added risks to the patients.
In patients with hepatic cirrhosis and ascites, sudden alterations of electrolyte balance may precipitate hepatic encephalopathy and coma. Treatment in such patients is best initiated in the hospital with small doses and careful monitoring of the patient's clinical status and electrolyte balance. Supplemental potassium and/or spironolactone may prevent hypokalemia and metabolic alkalosis in these patients.
In cats, dogs and guinea pigs, Bumex has been shown to produce ototoxicity. Since Bumex is about 40 to 60 times as potent as furosemide, it is anticipated that blood levels necessary to produce ototoxicity will rarely be achieved. The potential for ototoxicity increases with intravenous therapy, especially at high doses.
Patients allergic to sulfonamides may show hypersensitivity to Bumex.
**PRECAUTIONS:** Measure serum potassium periodically and add potassium supplements or potassium-sparing diuretics, if necessary. Periodic determinations of other electrolytes are advised in patients treated with high doses or for prolonged periods, particularly in those on low salt diets.
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Drugs with nephrotoxic potential and bumetanide should not be administered simultaneously.
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Probenecid should not be administered concurrently with Bumex.
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Interaction studies in humans have shown Bumex to have no effect on warfarin metabolism or on plasma prothrombin activity.
*Pregnancy*: Bumex should be given to a pregnant woman only if the potential benefit justifies the potential risk to the fetus.
Bumetanide may be excreted in breast milk.
*Pediatric Use*: Safety and effectiveness below age 18 not established.
**ADVERSE REACTIONS:** Muscle cramps, dizziness, hypotension, headache and nausea, and encephalopathy (in patients with preexisting liver disease).
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**Before prescribing, please consult complete product information, a summary of which follows:**
**Indications:** Management of anxiety disorders; short-term relief of anxiety symptoms, acute alcohol withdrawal symptoms, preoperative apprehension and anxiety. Usually not required for anxiety or tension associated with stress of everyday life. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.
**Contraindications:** Known hypersensitivity to the drug.
**Warnings:** Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
**Precautions:** In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.
**Adverse Reactions:** Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment, blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
**Usual Daily Dosage:** Individualize for maximum beneficial effects. *Oral—Adults:* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.*, severe states, 20 or 25 mg *t.i.d.* or *q.i.d.* *Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)
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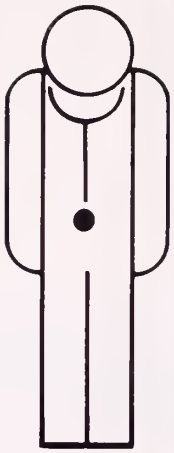

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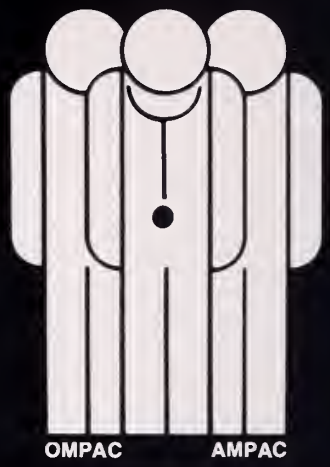


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# JOURNAL

## OKLAHOMA STATE MEDICAL ASSOCIATION

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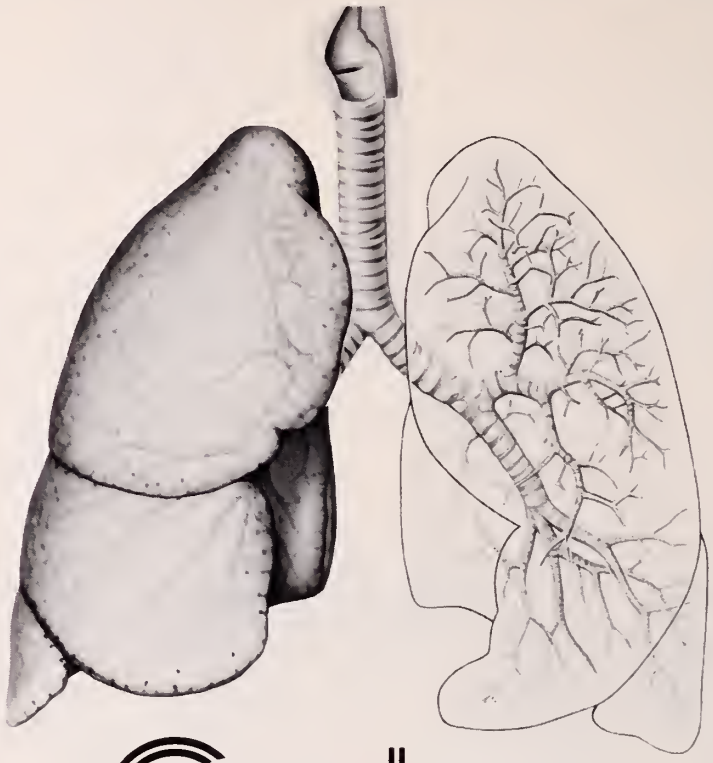

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A recent double-blind, placebo-controlled, crossover study in 138 hypertensive patients[2] revealed that INDERAL LA has a side effects profile unsurpassed by atenolol or metoprolol—which shows how well-tolerated once-daily INDERAL LA can be.

## Sole therapy or concomitant therapy? Fifty-nine percent of the time, INDERAL LA stood on its own.

The patients in the nationwide compliance trial were no different from yours. Generally when the antihypertensive regimen is complicated, compliance may become a problem. So, the effectiveness of INDERAL LA as once-daily monotherapy is a big plus. Of the remaining hypertensives in the program, 36% were controlled merely with the addition of a diuretic to INDERAL LA.

## For the noncompliant patients in your practice, INDERAL LA may well be the answer.

Almost 20,000 of the patients in the nationwide compliance trial were identified as having been noncompliant with their previous antihypertensive therapy. Their physicians reported that 88% showed improved compliance when placed on once-daily INDERAL LA.

# Control, comfort, and compliance

ONCE-DAILY
**INDERAL® LA**
(PROPRANOLOL HCl)
LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

# The one you know best keeps looking better

Please see next page for brief summary of prescribing information

# The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

**INDERAL® LA** brand of propranolol hydrochloride **(Long Acting Capsules)**

**DESCRIPTION.** Inderal LA is formulated to provide a sustained release of propranolol hydrochloride. Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.

**CLINICAL PHARMACOLOGY.** INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period. The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity.

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved. In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable.

**INDICATIONS AND USAGE. Hypertension:** INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

**Angina Pectoris Due to Coronary Atherosclerosis:** INDERAL LA is indicated for the long-term management of patients with angina pectoris.

**Migraine:** INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

**Hypertrophic Subaortic Stenosis:** INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

**CONTRAINDICATIONS.** INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

**WARNINGS.** CARDIAC FAILURE. Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

**Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)** — PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY. The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA. Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS. Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranolol.

**PRECAUTIONS.** *General.* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

*Clinical Laboratory Tests.* Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS. Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

*Carcinogenesis, Mutagenesis, Impairment of Fertility.* Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

*Pregnancy.* Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

*Nursing Mothers.* INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

*Pediatric Use.* Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS.** Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

*Cardiovascular.* bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

*Central Nervous System.* lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

*Gastrointestinal.* nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

*Allergic.* pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

*Respiratory.* bronchospasm.

*Hematologic.* agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

*Auto-Immune.* In extremely rare instances, systemic lupus erythematosus has been reported.

*Miscellaneous.* alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

**DOSAGE AND ADMINISTRATION.** INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION — *Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS — *Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained. Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE — *Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS — 80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE — At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

**REFERENCES:**


1. INDERAL LA National Compliance Evaluation Program. Data on file, Ayerst Laboratories.
2. Ravid M, Lang R, Jutrin I. The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily. *Arch Intern Med* 1985;145:1321-1323.


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New York, NY 10017

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"Living in the city is lonely enough... with herpes it's like solitary confinement."
ZOVIRAX®
(acyclovir)
CAPSULES
Prevent genital herpes recurrences month after month with daily therapy.
(In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.)
Please see last page of this advertisement for brief summary of prescribing information.

# ZOVIRAX® (acyclovir) CAPSULES

## Help free your patients from recurrences.

## Daily therapy

Coping with genital herpes is rarely easy. For some, the worst part is the pain and discomfort of frequent attacks —month after month, year after year. For others, the emotional burden presents a more difficult problem, leading to social isolation, anxiety, and diminished self-esteem.

## Prevent or reduce recurrences

Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

## Generally well tolerated

Daily therapy with ZOVIRAX CAPSULES is generally well tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/vomiting, vertigo, and arthralgia.

The physical and emotional difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episodes, as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRAX CAPSULES.

*Please see brief summary of prescribing information on next page.*

86-Z

# Prevent recurrences month after month*

# ZOVIRAX® (acyclovir) CAPSULES

Brief Summary

**INDICATIONS AND USAGE:** Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psychosocial difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

**First Episodes** (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

**Recurrent Episodes:**

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

**CONTRAINDICATIONS:** Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

**WARNINGS:** Zovirax Capsules are intended for oral ingestion only.

**PRECAUTIONS: General:** Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

**Drug Interactions:** Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the $F_1$ generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

**Pregnancy:** ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

**Nursing Mothers:** It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

**Pediatric Use:** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS — Short-Term Administration:** The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

**Long-Term Administration:** The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

**DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes:** One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

**Chronic suppressive therapy for recurrent disease:** One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

**Intermittent Therapy:** One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

**Patients With Acute or Chronic Renal Impairment:** One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance $\leq 10$ ml/min/1.73/m$^2$.

**HOW SUPPLIED:** Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200"- Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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*In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

Wellcome

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Research Triangle Park
North Carolina 27709

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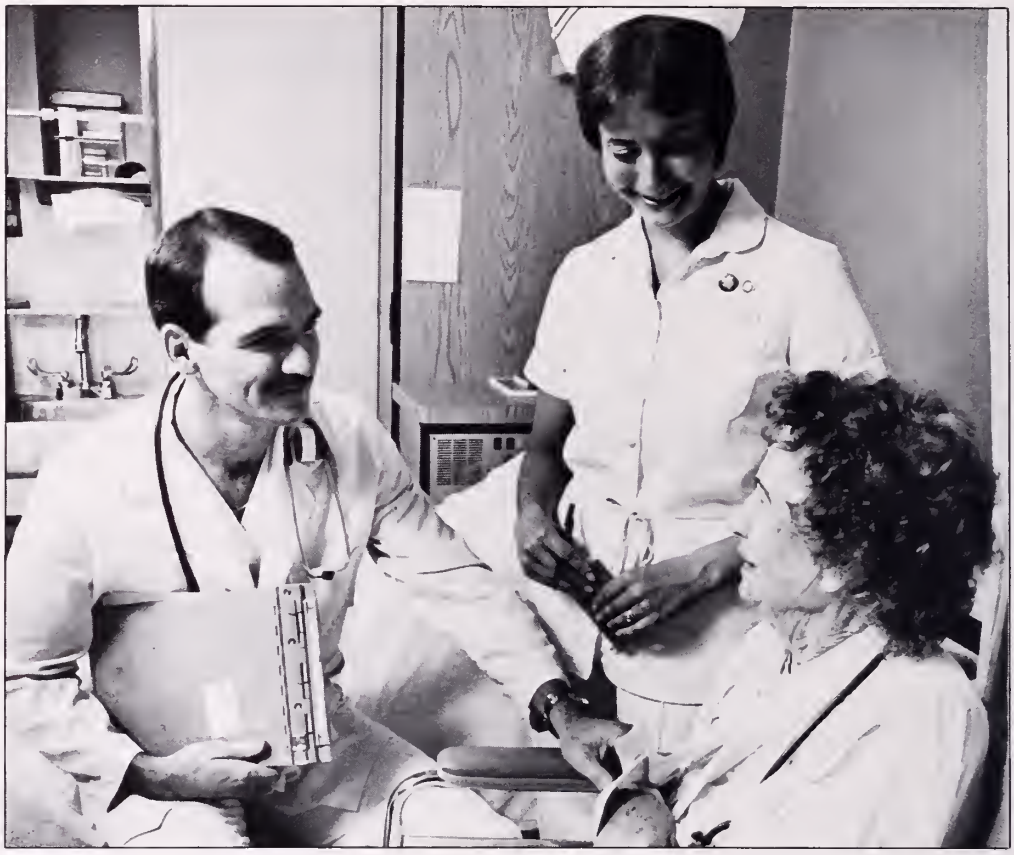
# She wants to go home . . .



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You demand quality service for your patients and Allied Nursing Care provides the best care, anywhere in Oklahoma.

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# A Winter Wonder Land

Did you ever wonder why the actual cost of a medical education has never been publicized? The investment of nine to thirteen years of prime youth; the value of the potential earnings of those years; the interest that sum could have earned; the cost of each year of study; the amount of interest owed on borrowed monies; the comparative risk of not being able to complete the course of study?

Did you ever wonder why the actual earnings of physicians have never been publicized? The physicians' incomes on an hourly basis, allowing time-and-a-half or double time for every hour worked in excess of forty hours per week, at nights and on holidays; the incomes of physicians who work in residencies, fellowships, and research assignments; the value of fringe benefits not applied to physicians' jobs; the net professional lifetime earnings of physicians?

Did you ever wonder why the contributions of physicians to hospitals are never publicized? The millions of physician hours contributed to hospitals as staff members, department chairpersons, chiefs of staff, committee members, committee chairpersons; in caring for patients who arrive in emergency rooms needing but not having a physician and being unable to pay for their care; participating in countless inspections, continuing education programs, fund drives, and public service programs — all without one cent of remuneration?

Did you ever wonder why the selective and capricious restrictions on physicians as compared to other professionals in our society are not publicized? Why is it that lawyers and engineers and accountants are not only permitted but expected to be paid for their time; why they can and do negotiate contracts with their clients; why they can, in most cases, determine their own fees; why is it when lawyers and judges are disciplined, their hearings are held in secret while physicians must be tried in meetings open to the public; why is it that the licensing and supervising agency for lawyers in this state consists of state employees on full-time salaries while the members of the Board of Medical Examiners serve voluntarily and, except for the secretary, without any compensation for their time and liability; why is the lawyers' licensing agency not required to contribute a cent of its revenues to the state while the Board of Medical Examiners must send to the state treasurer ten percent of its gross revenues?

Did you ever wonder why the credentials and salaries of the bureaucrats appointed to regulate physicians' practices are never publicized? Why our credentials must be displayed, available in files open to the public, and repeatedly verified; why our pay is frozen or decreased and generally exaggerated while their salaries as well as their numbers steadily and annually rise by amounts which are always defended and generally not made public?

Did you ever wonder why these issues are not publicized? Or who should publicize them? Or if some of the membership fees you send to your professional organizations should be spent for publicizing them?

Did you ever wonder why we never learn?

— *MRJ*

Dear Fellow Physicians:

1987 begins with little change in the current medicine-patient environment in which we work.

Pressures of liability fears remain. Problems with different economic methods of health care delivery continue. The frustrations of dedicated doctors trying to give good conscientious care to their patients are unablated, maybe even worse.

But there may be some light on the horizon. The public, our patients, is slowly beginning to realize the problems of medicine and especially to understand that these problems mean trouble to them, to their health, and to their well-being.

Yes, we are desperately in need of legislative help. We need reform in our malpractice liability situation. We need legislative protection so that we can more effectively police our own. And, we do need a more realistic appraisal by government and industry of the overall effects on health care when the main factor, often the only factor considered, is its cost and balance sheet.

However, we are not blameless. In fact, as a profession, our actions certainly explain some of the public's antagonism toward us.

We must better police ourselves. We must not be afraid to chastise the incompetent, the lazy, and the uncaring among us.

We must not play industry's game and must not allow ourselves to be caught up in wars of price-cutting, service withholding, and other forms of monetary savings that may be detrimental to the people we serve.

We must continue to do even better what we do best; that is to provide caring, compassionate, and skillful care to the best of our ability.

Norman L. Dunitz, MD

# Glycosylation... An Aid in Assessing Diabetic Control

DON P. WILSON, MD; J. L. HOROWITZ, MT; R. STRATTON, PhD; R. K. ENDRES, MD

**In individuals with diabetes mellitus, measurement of glycosylated proteins is a clinically useful and reliable method of assessing metabolic control.**

Reliable assessment of metabolic control is of paramount importance in individuals with diabetes mellitus. Traditional methods of evaluating blood glucose control, such as historical data, urine testing, and/or laboratory determination of fasting or random blood glucose levels, are both nonspecific and insensitive as indices of long-term control.[1] The presence of physical findings indicative of diabetic complications provides, at best, a cumulative index of chronic, poorly controlled diabetes. Recently, self-monitoring of blood glucose has added an important new tool to the armamentarium of the clinician. Yet, self-generated blood glucose determinations are highly dependent upon accurate performance and compliance. Recently we reported poor compliance and even fabricated test results in an alarming number of adolescents with insulin-dependent diabetes.[2] Similar discrepancies have been found in the test results of adults.[3] The ability to measure glycosylated proteins, especially hemoglobin (Hgb), now provides a reliable, efficient, and objective means of assessing metabolic control in individuals with diabetes mellitus.

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## Origin

When constantly exposed to glucose, proteins undergo a modification that allows the attachment of glucose, a process referred to as glycosylation or, more specifically, glucosylation. This reaction is nonenzymatically mediated and irreversible in the final ketoamine form. Glycosylation is a two-step process involving aldimine as an intermediate product. Aldimine, a Schiff base, is labile and easily reversible. Methods of determining glycosylated proteins that do not eliminate or exclude this labile fraction may result in falsely elevated values.

## Glycosylated Hemoglobin

Red blood cells are freely permeable to glucose; thus, hemoglobin is constantly exposed to the ambient glucose concentration. Since blood samples are readily accessible in clinical practice, hemoglobin has become the most common index of glycosylation. However, structural proteins as well as blood components undergo similar changes.

A blood specimen for determination of glycosylated hemoglobin may be obtained randomly without regard to time of day, recent meal ingestion, or illness. Assuming the labile fraction has been eliminated, recent or transient fluctuations in blood glucose should have no influence upon test results.

Hemoglobin A accounts for over 90% of total hemoglobin. Using electrophoresis or ion exchange chromatography, hemoglobin A can be separated into

four main subfractions designated hemoglobins $A_{1a1}$, $A_{1a2}$, $A_{1b}$, and $A_{1c}$. Hgb $A_{1c}$ is quantitatively the largest subfraction and can be specifically measured using selective separation techniques. However, because of the minor contributions of the remaining hemoglobin subfractions, many commercial assays measure glycosylation of all subfractions, reporting total glycosylated hemoglobin. Results are expressed as a percentage of the total hemoglobin. Thus, a glycosylated hemoglobin of 14%, for example, indicates that 14% of the total hemoglobin assayed has glucose irreversibly attached.

Values for glycosylated hemoglobin are thus dependent upon two major factors: (1) the average 120-day lifespan of the red blood cell, and (2) the mean blood glucose concentration. Factors that affect either variable may alter the degree of glycosylation present.

## Interpretation of Results

Glycosylated hemoglobin determination is an indirect measurement of mean blood glucose concentration. Each 1% change in glycosylated hemoglobin is approximately equal to a change of 25-35 mg/dl in the mean plasma glucose.[4]

Results from different laboratories are generally not comparable since no reference standard exists. In our laboratory, the range of values for nondiabetics is 4% to 8%. Arbitrarily, we have established the following ranges to aid clinical interpretation: 4% to 8% normal, 9% to 12% intermediate, >12% high. We, as others, have found that insulin-dependent individuals with Hgb $A_1$ values in the normal range are at a higher risk for hypoglycemia. Such individuals should be closely monitored and properly instructed in the treatment of symptomatic hypoglycemia.

## Assay Limitations

As with any laboratory determination, a variety of factors may result in spurious test results. Particularly when cation exchange chromatographic techniques are used, the clinician should be aware of the most common errors.

Improper specimen collection, transport, and storage can greatly affect test results. Whole blood specimens can be stored at 4°C for approximately one week without significant alteration. At temperatures of −70°C or colder, specimens are stable for several months.

False elevations in test results may result from adverse assay conditions (temperature, pH), failure to eliminate the labile fraction (aldimine), hyperlipidemia, hemoglobin variants (HgbF), drug or chemical modification of hemoglobin (aspirin, ethyl alcohol), anemia, or with increased red blood cell survival time (as after splenectomy).

A lowering of test values may be seen in individuals with alterations in the red blood cell pool (hemolytic process, recent transfusion), hemoglobin variants (HgbS or C), or variations in assay conditions (temperature, pH).

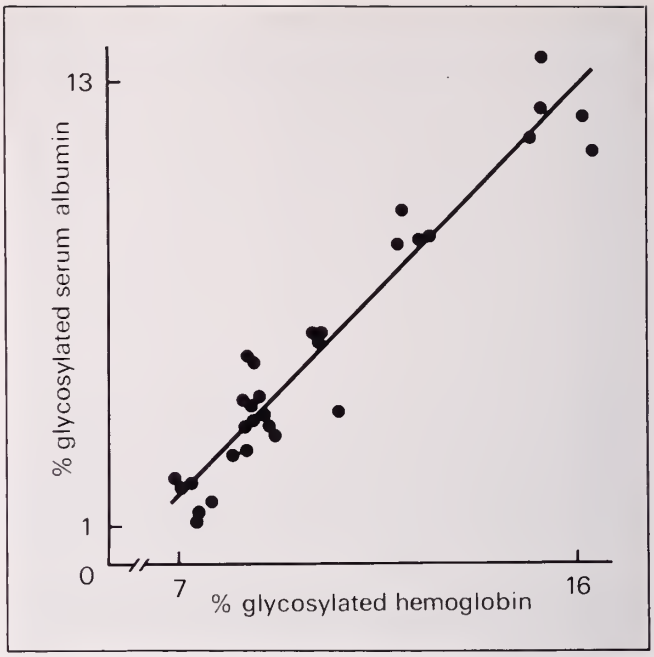


**Fig.** Correlation coefficient (Pearson's r), glycosylated serum albumin (%GSA), and glycosylated hemoglobin (%HbA1) from the same blood sample in 32 adolescents with insulin-dependent diabetes mellitus, r=.96, p<.0001.

## Blood Glucose Monitoring

Self-determination of blood glucose values in the fasting, pre- and post-prandial state has largely replaced urine testing for glucose in the daily management of insulin-dependent diabetes. Often, fasting blood glucose values haved been used as indices of metabolic control. Although such measurements are subject to individual variability, a strong correlation does exist between glycosylated hemoglobin and both fasting and two-hour post-prandial plasma glucose concentrations.[5] A similar relationship can be demonstrated between mean blood glucose values obtained from self-monitoring and glycosylated hemoglobin. Thus, while individual determinations of blood glucose yield specific information useful in altering the daily treatment regimen, the glycosylated hemoglobin level provides an average of the blood glucose over the preceding 6 to 8 weeks. Glycosylated hemoglobin values,

therefore, serve as more suitable biologic markers of metabolic control, over time, than do random blood glucose values.

Reliable short-term evaluation of metabolic control might be achieved by measuring glycosylation of other serum proteins.[6] Albumin, for example, has a much shorter survival in serum (half life 20 days) than does hemoglobin.[7] Simultaneous values for glycosylated serum albumin and glycosylated hemoglobin are highly correlated ($r = 0.96$) (Figure). Such short-term assessment of metabolic control has many potential applications in both clinical practice and research. During pregnancy, for example, there is a real need to maintain excellent diabetic control to decrease infant morbidity and mortality.

Recently we reported a decline in glycosylated serum albumin in diabetic adolescents with parallel improvements in physical fitness during a supervised eight-week exercise program.[8] No changes were seen in simultaneous glycosylated hemoglobin values. Apparently, glycosylated serum albumin may provide an objective marker, useful in deciding the benefit of therapeutic decisions much earlier than does glycosylated hemoglobin. Results may be a potential motivating factor for individuals participating in such a program.

## Diabetes Detection

The utility of the standard oral glucose tolerance test (OGTT) in detecting diabetes mellitus is limited by many factors. Age, nutritional status, diet, time of testing, and physical activity may all have a significant impact on test results. Measurement of glycosylated hemoglobin or serum proteins, alternatively, is not affected by these variables, thus offering a potential advantage as a method for diabetes detection. Both glycosylated hemoglobin and glycosylated serum proteins have shown an ability to clearly discriminate diabetic from nondiabetic individuals. Using commonly accepted scoring methods, Dods et al[9] found normal glycosylated hemoglobins in 37% to 64% of individuals considered diabetic by the OGTT. An impaired ability to handle glucose, however, can be present despite normal glycosylated hemoglobin and glycosylated serum proteins. Thus, while both glycosylated indices potentially may be reliable aids in diabetes detection, neither is suitable in determining the presence of "impaired glucose tolerance."

## Summary

Measurement of glycosylated serum proteins in individuals with diabetes mellitus is an accurate index of metabolic control over a period of time. Glycosylated hemoglobin estimates the mean blood glucose concentration over the preceding 6 to 8 weeks. Because of its shorter serum survival time, glycosylated albumin reflects the mean concentration over a shorter interval.[10] Although not suited for determining changes in the daily management regimen, results are helpful in setting goals for standards of self-care. Results between visits can be compared to provide valuable feedback.

Physicians should be aware of factors that may artificially alter test results. Periodic determination of glycosylated proteins, such as hemoglobin in individuals with diabetes mellitus, should provide a more objective assessment of metabolic control than has previously been possible.

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# Thyrotoxic Hypokalemic Periodic Paralysis: Report of Four Cases and Review of the Literature

(Second of two parts)

THOMAS D. TINKER, MD, and JERRY B. VANNATTA, MD

**Hyperthyroidism is associated with several neuromuscular disorders, one of which is periodic paralysis. Hypokalemia almost always accompanies the dramatic paralytic episodes which characterize this rare disorder, hence the name thyrotoxic hypokalemic periodic paralysis (TPP). Presented are four cases, including the first reported in a Vietnamese male and the first associated with lymphocytic thyroiditis. One (case 3) is of undetermined cause. A review of the literature is included, as well as a thorough discussion of clinical findings and pathophysiologic mechanisms. A comparison between TPP and familial periodic paralysis is made.**

A clinically useful classification for the periodic paralyses and one which serves as a differential diagnosis for thyrotoxic hypokalemic periodic paralysis (TPP) is presented in Table 1.

## Thyrotoxic vs Familial Periodic Paralysis

Similarities between thyrotoxic and familial hypokalemic periodic paralysis (TPP and FPP) are numerous. In both, a male preponderance exists, but the thyrotoxic variety shows a greater predilection (range 6:1 to 15:1) than does the familial (range 3:1 to 12:1). Though Engel,[8] in his 1961 review of the world literature on TPP, found the male:female ratio to be greater than 6:1, the 16 reported cases in Caucasians up to that time were all male, and of the 14 cases reported since that time, 13 have been male.

TPP is clinically and biochemically indistinguishable from FPP when the findings associated with hyperthyroidism are excluded. Factors which induce paralytic attacks are the same and include cold, trauma, infection, alcohol, emotional excitement, large meals of high carbohydrate or sodium content, strenuous exercise followed by rest, and prolonged sleep.[2] Iatrogenic induction occurs with ACTH, epinephrine, glucose, insulin, and corticosteroids such as fluorohydrocortisone.[2] A fall in potassium with concomitant decline in urine potassium excretion occurs, and there is no evidence of excessive potassium loss in the stool during attacks.

During the onset of paralysis, a constant positive arteriovenous difference in potassium is observed, implying that the drop in serum potassium is secondary to intracellular movement of potassium from the extracellular space rather than a total body potassium deficit.[17] In TPP the total body exchangeable potassium is normal.[17] Response to treatment of acute attacks is similar, in that resolution of paralysis usually occurs with administration of potassium salts.[2,19] The ease with which normokalemia is reestablished supports maldistribution of

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serum potassium rather than total body depletion.[19-22] Microscopically, the most characteristic change in affected muscles is vacuolation.[8,15] The exact origin of these vacuoles and their pathologic significance remains undetermined.

Fewer substantial differences exist between TPP and FPP. In TPP a family history is highly uncommon, whereas in FPP, as the name implies, a positive family history is the rule.[2] The age of onset varies considerably between the two. In FPP greater than 60% of reported cases are symptomatic before age 16 years, whereas in TPP, 85% become symptomatic between the ages of 20 and 39 years,[23] corresponding to the age incidence of thyrotoxicosis.[24]

In a recent retrospective study of TPP in Japan, looking at greater than 25,000 thyrotoxic patients, Hamada et al[25] found the peak incidence of TPP in males to be 20 to 29 years of age, with no patient less than 14 years old. They suggest that the occurrence of TPP in this age group may be related to male sexual maturity. A tabulation of the 13 cases of TPP in Caucasian males, appearing in the English literature after 1961, reveals that 69% of reported cases fall within the age range of 20 to 39 years. All four patients presented in this paper are in the age range of 20 to 29 years.

Response to thyroid extract illustrates the most significant difference between TPP and FPP. Giving thyroid extract to TPP patients results in increased frequency and severity of paralytic attacks.[4,26] On the other hand, FPP patients given thyroid extract experience an exacerbation of weakness when the extract is withdrawn but obtain relief when the extract is again administered.[8]

**Table 1. — Classification of Periodic Paralysis**

I. Primary periodic paralysis
- A. Familial hypokalemic periodic paralysis
- B. Hyperkalemic paralysis with or without myotonia
- C. Normokalemic periodic paralysis
- D. Paramyotonia congenita with myotonia and weakness on exposure to cold

II. Secondary periodic paralysis
- A. Thyrotoxic hypokalemic periodic paralysis
- B. Paralysis associated with other endocrinologic causes of hypokalemia, ie:
  1. Mineralocorticoid excess
  2. Renal tubular acidosis
  3. Diabetic ketoacidosis
  4. Licorice intoxication

## Inheritance

All primary periodic paralyses (Table 1) are transmitted by an autosomal dominant gene with the hypokalemic type showing reduced penetrance in females. Among these, the hypokalemic variety is the most common. All of the secondary periodic paralyses manifest clinically with hypokalemia, and among these, paralysis associated with thyrotoxicosis is the most common.

Historically, the absence of periodic paralysis in the family history was considered almost pathognomonic for TPP. However, data are in the literature which suggest that a familial component is demonstrable in a small percentage of patients with TPP.[7,27] McFadzean and Yeung[24] surmise that the susceptability of certain hyperthyroid patients to periodic paralysis may be genetically determined, but that the precise mode of inheritance is not yet identified. They and others[28] suggest that the basic defect in TPP patients becomes manifest only when challenged by thyrotoxicosis, but that the occurrence of paralytic attacks is not related to the duration or severity of the thyrotoxicosis. Indeed, McFadzean and Yeung[24] were able to isolate a subgroup of thyrotoxic males without a history of paralysis in whom attacks could be experimentally induced. They[24] initially defined the incidence of TPP among southern Chinese and later described two Chinese families in whom nine members of two successive generations developed TPP.[29] Seven of the nine were female, an exceedingly rare occurrence.

**Resolution of the paralysis usually occurs with administration of potassium salts.**

The only well-documented case of TPP occurring in a Caucasian female was reported in a 24-year-old whose father and uncle carried the diagnosis of familial periodic paralysis.[28]

Yeo et al,[30] in their controlled study of 21 unrelated Chinese males with TPP, discovered two HLA antigens (AX BW22 and AW19 B17) not present in patients without TPP. Although they suggest that these haplotypes may serve as genetic markers for

TPP, they caution that it remains to be seen if all thyrotoxic Oriental males with these haplotypes will go on to develop TPP or if paralytic attacks can be induced in these patients.

A recent study from Japan suggested that diet may influence the occurrence of periodic paralysis in hyperthyroid patients.[25] A persistent decline in prevalence of TPP in Japan during the preceding 10 years was noted. The authors conjectured that this decrease may have been linked to the westernization of the Japanese diet; since a larger proportion of their staple diet is now protein, carbohydrate intake (rice) is declining.

## Incidence

In the last 30 years, several studies have assessed the incidence of TPP in populations such as the Japanese and the Chinese. Reports by Okinaka et al[15] and Satoyoshi et al[23] cited incidences among thyrotoxic Japanese of 1.9% and 8.8%, respectively. In the thyrotoxic population of Okinaka et al, 8.2% of males and 0.4% of females evaluated developed hypokalemic periodic paralysis (HPP). Hamada et al[25] recently reported the results of a 12-year study examining almost 5,000 hyperthyroid males and found the incidence of periodic paralysis in this population to be 10.1%. In more than 20,000 female hyperthyroid patients, there were no cases of TPP. Okihiro and Beddow, in a study of TPP in Hawaii, found that 9 of 10 male patients with TPP were of Japanese ancestry, further supporting the disease's predilection for young adult Japanese males.[31]

Chen et al[32] reported the occurrence of periodic paralysis in Taiwan and found only one thyrotoxic case among the 28 cases studied. Twenty-six of the 28 patients were male, and 25 of the 28 cases were found to be idiopathic. The three cases in which specific etiologies were documented included periodic paralysis secondary to hyperthyroidism, a potassium-losing nephritis, and primary aldosteronism. Chen concluded that nonfamilial thyrotoxic periodic paralysis is more common in Japan, whereas nonfamilial idiopathic periodic paralysis is more common in Taiwan.

**TPP has been said to occur with greater frequency among Orientals.**

McFadzean and Yeung[39] reported TPP occurring in 1.8% of thyrotoxic southern Chinese (13% males and 0.17% females), but stated they had also observed its occurrence in northern Chinese. They mentioned two reports of single cases among Koreans and related that the Korean authors felt the true incidence of TPP was higher than was represented in the literature.[33,34]

As a result of Engel's report,[8] in which 90% of TPP cases cited in the world literature came from Japan, TPP has been said to occur typically with greater frequency among Orientals. Though no study designed to ascertain the incidence among Caucasians has been performed, it has been reported that in a single series of 63 consecutive cases of thyrotoxicosis (gender not specified), four white males (6%) had periodic paralysis.[35] Despite the failure of the study to report the ethnic origin(s) of the remaining 49 thyrotoxic patients, the data were presented in the English literature and, therefore, one might anticipate that the majority of patients were Caucasian. In any event, even if a small number of the remaining 49 unidentified patients were not Caucasian, the incidence of TPP among Caucasians in the study was at least 6%.

TPP has also been reported to occur in Filipinos,[15,36] a Puerto Rican,[19] Mexican Americans,[36] American Indians,[37] blacks,[38,39] a Latin American,[40] and a Cambodian.[41] Ours is the first case report of TPP in a Vietnamese male and the fourth and possibly fifth in American Indian males in the English literature. Anthropologic data reveal that American Indians[37] and Vietnamese may share a common ancestry with other Mongolians. If this supposition is correct, one would expect the incidence of periodic paralysis among thyrotoxic American Indians and Vietnamese to approximate 2%. For the present, it must suffice to alert physicians delivering medical care to these ethnic groups that a high incidence of TPP possibly exists among them. Bernard el al[36] attempted to promulgate the same type message to physicians in the Southwest in their report on TPP among Mexicans and Mexican Americans.

## Clinical Presentation

In the majority of cases, thyrotoxicosis precedes the development of paralysis, but paralysis may antedate the thyrotoxicosis.[36,39,42] Because it is not

uncommon for the patient's thyrotoxic state to present subclinically,[36,42,43] a high index of suspicion must be maintained if the diagnosis is to be made.

Prodromal symptoms including muscle aches and cramps in the affected extremities may accompany attacks of paralysis. Some patients may even complain of irritability and apprehension. These symptoms may be so characteristic that the astute patient will learn to recognize them as indicative of an incipient attack. If the patient is awake during an attack, he may exercise the muscle groups which are cramping and frequently abort the attack,[44] but development of weakness while exercising has been reported.[45]

**Attacks may have a rapid onset, within hours.**

More commonly, the patient awakens early with widespread paralysis. Attacks may have a rapid onset, within hours. The limbs and the limb girdles are predominantly affected, and weakness in the lower extremities usually precedes weakness in the upper. Symmetric limb involvement is most common with any combination of single or multiple extremities being affected, but asymmetric involvement has been reported.[24] Extensor muscles are usually more involved than flexors, as are proximal muscles more than distal. Attacks may range from mild weakness to flaccid paralysis; hyporeflexic to areflexic deep tendon reflexes occur more commonly. However, instances of normal[16,45,46] deep tendon reflexes during the height of paralytic episodes have been reported.

At least two cases in the literature describe neurologic abnormalities concurrent with paralytic episodes, suggesting a corticospinal tract lesion.[16,22] In such an instance, lower motor neuron symptoms may resolve with correction of the hypokalemia, but upper motor neuron signs may not resolve until the patient becomes euthyroid.[22]

These neurologic phenomena, however, remain unexplained. These findings may cloud the clinical picture and obscure the correct diagnosis. Again, the necessity of a high index of suspicion for TPP is apparent. Involvement of extraocular muscles, as well as the muscles of phonation, deglutition, and sphincter control is rare. Compromise of sensory function and impairment of mental faculties or consciousness does not occur even in the severest of attacks.[2] Episodes last from one hour to four days, the average being 6 to 24 hours.

In the first reported case of TPP from the United Kingdom, all paralytic attacks occurred on the weekends.[46] This observation concurs with a tendency for attacks, as noted by Streeten, to appear on the same day of the week, perhaps in relation to eating and drinking habits.[47] In most cases, however, occurrence is erratic. The muscles paralyzed last recover first. Recovery is usually obtained within 3 to 12 hours, though some residual stiffness is often present. Between attacks, apart from the manifestations of hyperthyroidism, the patient is asymptomatic.

Rarely, the respiratory muscles are affected, and when this occurs, it may prove fatal. Among all forms of HPP, approximately 40 fatal cases have been mentioned in the literature.[35] Causes of death are cardiac failure or respiratory failure resulting from paralysis of the diaphragmatic and accessory muscles of respiration.[5,38]

## Electrocardiographic Findings

Ee and Cheah[48] found typical features of hypokalemia (U waves in Leads II, V-2, V-3, and V-4, progressive flattening of T waves, and depression of the ST segment) in all patients with TPP whose serum potassium levels were less than or equal to 2.8 mmol/liter. However, they did not observe certain electrocardiographic abnormalities associated with TPP which have appeared in case reports elsewhere. These include prolonged PR intervals,[20,42,49] supraventricular extrasystoles,[41] ventricular extrasystoles,[19,41] and intraventricular blocks.[24] Significant findings in this study[48] were the types of cardiac arrhythmias observed. Sinus arrest, occurring in two patients with serum potassium levels of 1.9 mmol/liter, was reported for the first time in hypokalemia without the added effect of digitalis. Second-degree atrioventricular block, occurring in three patients with serum potassium levels less than 2.1 mmol/liter, was also reported for the first time. The authors concluded that the occurrence of such potentially dangerous arrhythmias suggests that hypokalemia in TPP should be promptly corrected. It is presently felt that induction of paralysis has no therapeutic merit and, therefore, should only be attempted in an experimen-

tal setting under continuous cardiac monitoring with potassium readily available for intravenous injection.

Periodic paralysis with arrhythmia is a rare subgroup of HPP. Fisher[19] recently reported a patient with TPP in whom ventricular fibrillation and complex ventricular arrhythmia during paralytic attacks responded to potassium replacement. Since serious ventricular arrhythmias are often refractory to conventional antiarrhythmic agents, early evaluation of serum electrolyte levels in the patient with arrhythmia is important. Recognition of compartmentalization of potassium (ie, by finding an inappropriately elevated serum potassium level following a brief period of intravenous potassium replacement) rather than total body depletion, may lead to more prompt diagnosis and treatment of a potentially fatal illness.

## Treatment

In the acute management of paralysis, the patient is usually treated adequately by administering oral potassium chloride, careful observation, and serial serum potassium determinations. Potassium chloride should be given orally if possible. The average dose required per paralytic attack is 130mEq, in divided doses, usually over a period of 12 to 24 hours. An unsweetened aqueous solution[50] should be given because dilutents used to administer potassium salts by mouth, such as glucose and saline, may actually cause an initial worsening of hypokalemia.[51] Since the patient's muscles will contribute potassium to the extracellular fluid compartment at a rate up to 15 mEq/hour during recovery,[43] potassium should be given orally whenever possible because of the potential hazards which accompany intravenous potassium.[51] If the patient shows no sign of recovery within 3 to 4 hours, the dose may be repeated.[50] Administration of intravenous potassium is necessary only when the paralytic attack is complicated. It is indicated when the patient is unable to tolerate oral medications, there is cardiac dysrhythmia, or there is respiratory distress secondary to compromise of the diaphragmatic and accessory muscles of respiration. The recommended dosage of intravenous potassium is 60 mEq given by slow intravenous infusion.[51]

Recurrence of attacks in TPP is not uniformly prevented with prophylactic potassium administration.[37,52] Though Griggs[53] and others[54,55] noted dramatic improvement in FPP patients treated with acetazolamide, beneficial effects in TPP patients treated with the same agent have not been observed.[56] To the contrary, one study suggests that acetazolamide may aggravate TPP.[57]

Two studies comparing different approaches to medical management of TPP patients reveal that marked improvement of thyrotoxic symptoms as well as relief of paralytic episodes are obtained with the use of propranolol.[37,57] Propranolol's mode of action is felt to be blockade of beta-adrenergic receptors. Therefore, these authors felt that episodic muscle paralysis seen with thyrotoxicosis may be related to the hyperadrenergic state. In Yeung and Tse's study,[57] 71% (5 to 7) of patients given propranolol experienced partial or complete protection from induced paralysis. When the 5 patients were treated with propranolol and carbimazole, none experienced attacks during eight weeks of observation. Prior to the institution of propranolol therapy, the number of attacks in the same period ranged from 4 to 28. These and other authors are careful to point out that propranolol, like other therapeutic modalities such as potassium salts and spironolactone, afford protection in only a proportion of patients. However, they believe propranolol is a valuable adjunct in managing thyrotoxicosis in the Mongolian peoples, a group with a propensity to develop periodic paralysis.

In the vast majority of TPP cases reported in the literature, paralysis occurs only when the patient is thyrotoxic, and spontaneous attacks not only disappear[24,37] but the susceptibility to induced attacks disappears when the patient becomes euthyroid.[24,58] Treatment of TPP is effective and the euthyroid status can be reestablished with medical or radioactive suppression or surgical ablation of the thyroid.

**The patient is usually treated by administering oral potassium chloride.**

Numerous studies have shown that when the patient is euthyroid, even induction of hypokalemia does not precipitate a paralytic attack.[15,20,24,57] This finding suggests that the basic defect in TPP is latent

and that it is unmasked in the thyrotoxic state. After treatment, the malady persists though, for paralytic attacks may reappear if hyperthyroidism recurs. This phenomenon, which occurs in patients with resurgence of endogenous hyperthyroidism, has been reported in a patient who voluntarily took excessive doses of liothyronine and became thyrotoxic following seven years of normal thyroid function after subtotal thyroidectomy for thyrotoxicosis.[58] However, the literature contains two well-documented case reports[49,59] where episodes of paralysis persisted in patients who were clinically and biochemically euthyroid.

It has been proposed that a multiplicity of factors influence the clinical syndrome of TPP and that antithyroid measures may not be as rapidly or as uniformly curative as was once believed.[49] These two cases of TPP represent a small minority of the total number in the literature, so it is concluded that, based upon the majority of data available, propranolol and avoidance of strenuous exercise and high carbohydrate meals provide the best approach to medical management of a patient as he awaits definitive therapy, ie, resumption of the euthyroid state. Patients may experience significant alleviation of symptoms with propranolol but, in general, will not be freed from disability until specific antithyroid treatment is successful.[46]

Reports of the Jodbasedow phenomenon, associated with TPP, have appeared in the literature.[21,23] One case is of particular interest because the patient was clinically and biochemically euthyroid but, because he had a nodular thyroid, iodine suppression of the goiter was attempted.[21] He subsequently developed TPP. The authors point out that multinodular goiter may be a predisposing factor for inciting hyperthyroidism and that development of TPP is not dependent on a specific cause of thyrotoxicosis, such as Graves disease. Indeed, the development of TPP has been reported in patients taking thyroid extract to lose weight, suggesting that they possess the same defect responsible for periodic paralysis secondary to endogenous thyrotoxicosis.[60] In older patients, whose physiologic adaptability is impaired, the combination of undiagnosed TPP and thyrotoxicosis may have severe consequences.

## Pathophysiology

The pathophysiologic mechanisms operative in TPP remain an enigma. A survey of the literature corroborates the fact that no single therapeutic measure or method of induction is universally applicable among patients with TPP. Based on this finding, it has been proposed that several mechanisms may be responsible for the onset of paralysis, but that only one exists in any given patient.[20] Myriad theories postulating the mechanisms responsible for periodic paralysis in patients with TPP have been proposed. Among these, the periodic paralysis has been attributed to abnormalities in potassium kinetics,[17,57,61] a disorder of muscle membrane excitability,[62] aberrations in calcium kinetics,[9,16,18] synergism between hypokalemia and hypophosphatemia,[63] increased secretion of aldosterone or an aldosteronelike substance,[20,64] abnormal glycogen deposition,[10,12,65,66]

**. . . Episodic muscle paralysis . . . may be related to the hyperadrenergic state.**

abnormal insulin secretion,[57,67] and hyperactivity of the sympathetic nervous system.[33,57] Though none of these postulates explains fully the recurrent paralysis in patients with TPP, those proposing the involvement of potassium, calcium, and the hyperadrenergic state of the sympathetic nervous system are the most promising to date.

In TPP the serum potassium is usually low during episodes of paralysis. Several studies[17,61,68] have demonstrated that the movement of potassium into muscle cells accounts for the hypokalemia usually observed during attacks in periodic paralysis. However, cases have been reported in which the patient with TPP remained normokalemic throughout the paralytic attacks.[24,45,59] Likewise, accounts have told of patients who have regained strength in the face of persistent, significant hypokalemia.[15,41] Having noted those observations, it appears that alterations in serum potassium levels alone cannot fully explain the episodic bouts of paralysis characteristic of TPP. Potassium kinetics are a component of the paralytic episode but may not mediate it.[12]

The reduction in evoked action potential amplitude during paralytic attacks indicates that a disorder of muscle membrane excitability exists.[62] The fundamental problem, a failure of the muscle membrane to depolarize, implies the resultant

paralysis is due to inadequate spread of excitation.[43] The diminished spread of excitation is believed, by some, to be due to aberrations in the inward spread of excitation into the myofibril, but the exact mechanism is not yet understood. Schutta and Armitage[16] demonstrated granularity in the sarcoplasmic reticulum cisterns in a patient with TPP and attributed it to calcium deposition. These granular deposits may interfere with calcium uptake by the sarcoplasmic reticulum following muscle contraction.

Subsequent studies have supported the possible role of calcium in the pathogenesis of periodic paralysis. Au and Yeung[9] corroborated this hypothesis by showing that the activity of the calcium pump during paralysis is decreased in patients with TPP. They suggest that "the unmasking effect of thyrotoxicosis may be due to its uncoupling action of oxidative phosphorylation, thus depleting the supply of ATP which serves as an energy source for the active transport system of calcium across the membrane of the sarcoplasmic reticulum. A decrease in activity of the calcium pump would affect relaxation and hence the next contraction with the ultimate precipitation of paralysis." Takagi et al,[18] in like manner, noted depression in velocity and capacity of sarcoplasmic reticulum calcium transport during paresis in a patient with TPP and observed a diminution of all sarcoplasmic reticulum functions. They state that "even if the sarcoplasmic reticulum change is a secondary phenomenon, it could be important in the genesis of paralysis; if the sarcoplasmic reticulum is unable to release calcium, excitation-contraction coupling fails and the muscle cannot contract."[18]

**Both endogenous and exogenous causes . . . have been linked with TPP.**

## Summary

The four cases presented illustrate typical clinical findings in thyrotoxic hypokalemic periodic paralysis. The first case of TPP in a Vietnamese male and the fourth and possibly fifth in American Indian males are reported. Because these groups have alleged ancestral connections with Mongolians, known to have a high incidence of TPP, physicians providing health care to these groups need to be aware that an increased incidence of TPP possibly exists among them also.

Both endogenous and exogenous causes of hyperthyroidism have been linked with TPP. The Vietnamese male presented in this report demonstrates, for the first time, the concomitant occurrence of lymphocytic thyroiditis with spontaneously resolving hyperthyroidism and thyrotoxic hypokalemic periodic paralysis. This new combination reillustrates that the patient with an appropriate genetic predisposition may episodically manifest hypokalemic paralysis whenever and however he becomes thyrotoxic.

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# Adie's Syndrome: Report of a Case

JOE D. HAINES, JR., MD

**Adie's syndrome, a dilatation of the patient's pupil, often appears without other symptoms and may frequently be overlooked. Its cause remains unknown.**

A nineteen-year-old white woman complained of a dilated left pupil of at least two months duration. This was accompanied by some blurring of vision, especially noticeable at night. Her friends had recently called attention to the condition and commented on her unequal pupils. She had no other symptoms other than an occasional frontal headache.

The patient reported no trauma to the eye or any significant medical history. She was on no medications and did not use alcohol or drugs.

On examination the patient was found to have anisocoria, with dilatation of the left pupil. The left pupil constricted very slowly in response to light and near fixation, while the right pupil reacted briskly to both light and near fixation. Visual fields and funduscopic findings were normal bilaterally.

Neurological examination revealed entirely absent knee and ankle jerk reflexes. Other findings of the neurological examination were within normal limits. A diagnosis of Adie's syndrome was made, and the patient was referred for further evaluation by an ophthalmologist/neurologist.

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## Discussion

In a classic 1932 paper, W. J. Adie described a syndrome in which a tonic pupillary reaction is associated with the absence of one or more tendon reflexes.[1] As later described by Walton, the typical case of Adie's syndrome has a sudden onset in a young woman often in her third decade of life. She or her friends notice a large pupil or she experiences a sudden mistiness of vision in one eye. Then ankle, knee, and deep tendon reflexes in the arms diminish in that order of frequency.[2]

Somewhat over a thousand cases of Adie's syndrome have been reported in the literature; however, it is doubtful that the true incidence will ever be known.[3] Often the syndrome produces no symptoms, or the symptoms are mild or ignored, and it may be discovered only during routine examinations.

The cause of the disorder is completely unknown. Various investigators have observed the condition following orbital trauma, in association with bronchial carcinoma presenting with a myasthenic syndrome, and following viral infections such as varicella, encephalitis, and herpes zoster ophthalmicus.[4] Cases which have come to autopsy suggest that degeneration of the ciliary ganglia of the affected eye is responsible for the pupillary abnormality. Neuronal degeneration of the dorsal root ganglia has also been discovered in these patients, which

would theoretically account for the areflexia.[5,6] Loewenfeld and Thompson have proposed the theory that damage to the ciliary ganglia is followed by imperfect reinnervation of the iris sphincters. The denervation causes the sphincter muscle to exhibit a cholinergic supersensitivity.[7] This supersensitivity can be employed as a clinical test for Adie's syndrome by the application of a miotic (pilocarpine), which can provoke an intense pupillary contraction in the Adie's pupil of some patients. The reliability of this test has recently been questioned, however.

Once thought to be a benign disorder, Adie's syndrome has been reported to show worsening of the sphincter palsy in some patients, and involvement of both eyes in certain instances. Patients with Adie's syndrome often experience problems with accommodation in the affected eye. Therefore, these patients should be evaluated for binocular functions as well as accommodative abilities. Providing corrective lenses to minimize accommodative imbalance is helpful in many patients. [8]

Various concentrations of miotics such as pilocarpine have been found to reduce anisocoria and improve vision in selected patients. The particular drug and its concentration must be selected according to the patient's individual needs, due to the variability of actual denervation in Adie's synrome.[9]

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## Coming in March . . .

In production for March are an article on enteral tube feeding and an overview of Legionnaires' disease. Also scheduled is the next story in the JOURNAL's Leaders in Medicine series and reaction to "The Ubiquitous Pharmaceutical Representative," published in December.

## News from the Oklahoma State Department of Health

# Health Promotion for Older People

The number of persons age 65 years and older is increasing at a rate twice that for any other age group in society. It is estimated that 210 Americans reach 100 years of age each week.

Studies indicate that health is among the chief concerns of older people. And, communicating health information is a major challenge being addressed by the Oklahoma State Department of Health.

A document prepared for the Department of Health and Human Services entitled *Aging and Health Promotion: Market Research for Public Education*, suggests health information can be effectively relayed to older people through various media channels. For example, older people are heavy viewers of television and watch television with greater frequency than do other age groups. Their viewing patterns are particularly high during the daytime hours when public service announcements are most likely to be aired.

Older people are also heavy readers of newspapers, which suggests the value of newspaper columns and articles as tools for disseminating health information. Newspaper magazine supplements frequently feature health issues and are widely read by older individuals.

Various news magazines, including *Time*, *Newsweek*, and *U.S. News & World Report*, offer another source of information for many older individuals. Certain specialty magazines are also widely read by older people. One magazine of particular interest is *Prevention;* older people form the bulk of its readership.

The document also suggests that health messages should be relevant to older people and simply presented. For instance, data show that 81% of men 65 and older are married, while only 43% of women in that age group are married. Many have a household income of less than $10,000 a year.

When planning a health promotion campaign for older people, it is important to use multiple channels of communication. This multiple channel strategy coupled with interpersonal communication can strengthen the success of a campaign to promote the health of older Oklahomans.

| DISEASE | November 1986 | TOTAL TO DATE | | |
|---|---|---|---|---|
| | | This Year | Last Year | 5 Yr. Avg. |
| AMEBIASIS | 0 | 7 | 14 | 14 |
| CAMPYLOBACTER INFECTIONS | 14 | 246 | 286 | — |
| ENCEPHALITIS, INFECTIOUS | 1 | 21 | 26 | 29 |
| GIARDIA INFECTIONS | 19 | 225 | 306 | — |
| GONORRHEA (Use ODH Form 228) | 847 | 11600 | 12052 | 13584 |
| HAEMOPHILUS INFLUENZAE INVASIVE DISEASE | 23 | 218 | 229 | — |
| HEPATITIS A | 20 | 321 | 426 | 525 |
| HEPATITIS B | 23 | 194 | 211 | 246 |
| HEPATITIS, NON-A NON-B | 9 | 59 | 66 | — |
| HEPATITIS UNSPECIFIED | 6 | 46 | 77 | 161 |
| MEASLES (RUBEOLA) | 0 | 39 | 1 | 9 |
| MENINGITIS, ASEPTIC | 11 | 128 | 140 | 182 |
| MENINGITIS, BACTERIAL (non-meningococcal, non H. Influenzae) | 5 | 70 | 59 | 51 |
| MENINGOCOCCAL INFECTIONS | 4 | 73 | 68 | 55 |
| PERTUSSIS | 2 | 29 | 28 | 32 |
| RABIES (Animal) | 1 | 58 | 103 | 141 |
| ROCKY MOUNTAIN SPOTTED FEVER | 3 | 103 | 93 | 123 |
| RUBELLA | 0 | 0 | 1 | 1 |
| SALMONELLA INFECTIONS | 29 | 451 | 423 | 441 |
| SHIGELLA INFECTIONS | 17 | 285 | 256 | 315 |
| SYPHILIS (Use ODH Form 228) | 17 | 151 | 192 | 188 |
| TETANUS | 0 | 1 | 1 | 1 |
| TUBERCULOSIS | 20 | 237 | 231 | 263 |
| TULAREMIA | 1 | 12 | 18 | 27 |
| TYPHOID FEVER | 0 | 2 | 2 | 3 |

| Diseases of Low Frequency | Total to Date This Year |
|---|---|
| ACQUIRED IMMUNE DEFICIENCY SYNDROME | 40 |
| BRUCELLOSIS | 0 |
| LEGIONNAIRES DISEASE | 18 |
| MALARIA | 12 |
| REYE SYNDROME | 5 |
| TOXIC SHOCK SYNDROME | 29 |

# He's Always Been There . . .

DEENA CANVASSER

Several months ago, at a surprise party celebrating his thirty-four years in practice, Tulsa obstetrician Eugene S. Cohen, MD, received this note from his daughter.

It is hard for me to believe that I am old enough to have a father who has been practicing medicine for 34 years. I tell myself I am not that old but I am. And I too was there at the beginning, although not cognizant of the fact. I was one of the reasons you kept working as hard as you have through the years, although not the only reason. Because I know that even if you had not had the impetus to be financially successful, you still would have strived to have been the best you could be. Thank G-d you had that drive. Thank G-d you had that dream because you have helped make the lives of hundreds of women worth living as well as those lives more comfortable to live.

No one can imagine what it must be like behind that mask, watching with bated breath the arrival of a newborn baby. To witness that miracle in awe and ecstasy, but also in fear and panic, until all can breathe a sigh of relief and say a silent prayer of thanks. You have been there for so many, in times of joy and happiness, in times of sorrow and pain.

I remember the little building on Peoria behind Grandpa's grocery store. Something green stands out in my mind. Did you have a green examining room? I remember the move to Lewis — the two examining rooms and how I used to scurry between the two. You were afraid your patients might be embarrassed by having your 13-year-old daughter there. I wasn't there for all the moves. I can't even remember the different floors you were on. I only remember the last move. How proud you were to walk me through those offices. Who would have imagined you would have come so far — from one doctor to 5, from 1 nurse to 14? But as proud as you were to show off your new offices to us, I don't think you have realized how proud we have all been of you throughout the years. Most parents watch their children grow to maturity with pride and admiration. I, the child, have watched you with pride, respect and affection.

**I have watched you with pride, respect and affection.**

I think the most difficult word I ever had trouble saying was *obstetrician*. But I was bound and determined to pronounce that word correctly. I was so proud. My dad was a doctor. But he was no ordinary physician who merely practiced his profession. He is a man who lives his profession. Every fiber of his

being is dedicated to the highest invincible principles of practicing the finest medicine there is. The goals and aspirations and principles you have set for yourself are far beyond the normal expectations of a man's abilities. Few could ever hope to attain such lofty heights. But you have practiced your profession with a profound dedication to these principles, never faltering, never compromising, never sacrificing your beliefs for convenience or urgency. You have lived your life according to your intense beliefs that above all else honesty and truth are not ever to be jeopardized, and compassion and caring for another one's own physical and emotional needs are the utmost of your concerns. I've heard your feet come scuffling down the hall after hours of an exhausting operation only to have to drag yourself out of bed again after another call from the hospital. I've seen the disappointment on yours and mother's faces after you have had to miss an important event or by the time you got home all three of us have had the 3-day measles. When people used to say to me, "It must be hard having your dad gone so much," I remember saying, "But he's always been there when we've needed him." The times you were with us were so special that I can't remember the times you weren't.

This tribute is also a testimonial to mother, for what you weren't able to provide for us in terms of emotional or physical support, mother had to take over and do double duty. Whether it was something involving us or just being alone at a party you couldn't attend, she was alone a good portion of the time. Your dedication to your livelihood, her dedication to you, and both your commitments to raising a loving and caring family have left indelible impressions on my mind. It is truly a hard act to follow. I don't know but I truly doubt that my children will have the kind of respect and admiration for me that your children have for both of you. I am not that self-sacrificing or that giving an individual. I too have my own needs. I know my children love me, but I don't know if my children will like me as much as all of us do you. But I do know my children share in that respect and esteem for their grandparents and understand the very special and unique human beings you are. And for that I am grateful. What better role models could I ever want for those I hold so dear. May G-d bless you with good health and happiness all the days of your lives. May you continue to enjoy and love what you are doing, for there are too many who enjoy and love you. May your patients always hear those footsteps down that hospital corridor, for only then will they rest reassured — Dr Cohen is here.

1987 OSMA
ANNUAL MEETING
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**The Select Committee** on Insurance Rates and Tort Claims met at the state capitol on January 8, 1987, to continue their efforts toward reform of the state's insurance and tort claim structure. Among those in attendance, shown here from left to right around the table, were Rep James E. Hamilton, D-Poteau; Rep Loyd Benson, D-Frederick; Sen Jerry L. Smith, R-Tulsa; Sen Bob Cullison, D-Skiatook; Rep A. C. Holden, D-Dewey; and attorney Larry Tarwater, Oklahoma City. Behind them, center, are OSMA Executive Director David Bickham and Associate Director Robert W. Baker. (More pictures on p 90.)

*"Effective vaccine is still years off"*

# AIDS dominates medical news in 1986 as cases double

AIDS — the effort to better understand how it ravages the body and the struggle to care for its ever-increasing number of victims — dominated medicine in 1986.

This past year, however, may be best remembered not as one of scientific breakthrough, says AMA Executive Vice President James H. Sammons, but as the time when health authorities officially and emphatically extended warnings about the disease to the general public.

"AIDS dominated the public health and infectious disease fields in 1986, but concern about this epidemic really has spread throughout medicine," he said. "This was the year that it became clear that AIDS is not a disease that just affects 'high-risk groups' but has implications for the community as a whole."

One expression of this concern came from the Institute of Medicine of the National Academy of Sciences, which called for a $2 billion AIDS education, public health campaign and expanded research effort. Underscoring the point was a straight-talk report from Surgeon General C. Everett Koop, who called AIDS "a major public health issue" and urged that education about it — including its modes of transmission — begin at an early age, both in school and at home.

"At the beginning of the AIDS epidemic, many Americans had little sympathy for people with AIDS," Koop wrote in the preface of his report, which was reprinted in the *Journal of the American Medical Association (JAMA)*. "Let us put those feelings behind us. We are fighting a disease, not people.

"The country must face this epidemic as a unified society," he said. "It is the responsibility of every citizen to be informed about AIDS and to exercise the appropriate preventive measures."

(continued)

**At the meeting** of the Select Committee on Insurance Rates and Tort Claims are, left to right, Don Blair, executive director of Oklahomans Against Lawsuit Abuse; Bob Schacher, general counsel to the Oklahoma Hospital Association; Rep John W. Bumpus, MD, R-Bethany; and David Bickham, executive director of the Oklahoma State Medical Association.

## AIDS dominates (continued)

The number of AIDS cases in the US was estimated at about 30,000 by year's end, double that of a year earlier, with up to 1.7 million people estimated to be infected with the human immunodeficiency virus (HIV). Although most victims are homosexual and bisexual men and intravenous drug users of both sexes, "heterosexual transmission is expected to account for an increasing proportion of those who become infected with the AIDS virus in the future," Koop wrote.

By the end of 1991, a decade after the first US cases were reported, the AIDS caseload is expected to reach 270,000 and the death toll 179,000, Koop said. During 1991 alone, he said, an estimated 145,000 AIDS patients will need health and support services expected to cost up to $16 billion. In short, AIDS' impact on society "is and will continue to be devastating," he wrote.

Despite importance advances in analyzing some of the basic ways in which HIV behaves, prospects for an AIDS vaccine brightened only slightly in 1986. "The more we learn about this virus, the more unpredictable it seems," Sammons notes. "Most experts still suggest an effective vaccine is still years off."

Some hope was generated by the finding that the drug azidothymidine (AZT) appears to stop the advance of the virus in patients with Kaposi's sarcoma, a skin cancer that is one of the primary symptoms of AIDS. Long-term prospects for this or other anti-AIDS drugs remain uncertain, however.

Other major developments in the year in medicine:

- The widening debate over drug use and the accuracy and effectiveness of mandatory, random urine screening.
- Government approval of interferon to treat hairy cell leukemia and a monoclonal antibody to treat kidney transplant rejection — the first of a new generation of such "biological response modifiers" released for therapeutic use.
- Word that the risk of coronary heart disease appears to be associated with far lower levels of cholesterol than earlier thought, and that the relationship between cholesterol and premature death from heart disease is a continuous, graded one posing a risk for a majority of middle-aged American men.
- Continued use of the artificial heart as a "bridge" for patients awaiting a transplant but a lull in its use as a permanent implant due to complications.
- Advances in efforts to pinpoint the genetic basis of disease as a first step toward better diagnosis and treatment, including isolation of the gene responsible for a form of muscular dystrophy and isolation of the first anti-oncogene.
- Widening debate over withholding life support for the hopelessly ill, fueled by an opinion from the AMA's Council on Ethical and Judicial Affairs that it is ethical to withhold fluids and nourishment from patients in a chronic vegetative state with no hope for recovery.

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*AMA requested clarification*

# Justice Dept says peer review not violation of antitrust law

The US Department of Justice has affirmed that good-faith peer review conducted by physicians on hospital medical staffs does not violate antitrust laws, according to a story in the December 19 issue of *American Medical News*.

*American Medical News* reports that a letter from the department to the American Medical Association says the "greatest potential of peer review is its ability to foster the basic goals of the antitrust laws in the health industry — the efficient delivery of quality services in a comprehensive marketplace."

The letter further states that "a hospital may legitimately decide to deny privileges to an incompetent practitioner, based on good faith peer review conducted by the medical staff. Although the denial of privileges might make it difficult or even impossible for the practitioner to engage in his profession, such a denial does not impair competition."

AMA Executive Vice President James H. Sammons, MD, said the letter came in response to the AMA's request that the Justice Department make clear its position with respect to peer review proceedings and antitrust law.

Although a $2.1 million jury verdict in an Oregon antitrust case has since been overturned, Sammons said this legal action had a chilling effect on doctors involved in the peer review process.

"The Justice Department letter is not binding in court," Sammons said, "but it is significant for its authoritative statement on the government's policy of promoting peer review and shielding peer reviewers from liability. More importantly, it encourages the AMA and other medical societies to continue to strengthen our commitment to strong peer review activities designed to provide patients with only the best care from competent physicians."

*FMG certification also discussed*

# Road to success in medicine now littered with obstacles

Young medical graduates face greater career risks than their predecessors have because of rapidly developing technology and changes in economic policies, according to two articles in the *Journal of the American Medical Association (JAMA)*. A third report describes factors influencing certification of foreign medical graduates (FMGs) in the United States.

"Historically, a physician had many different but clearly marked paths to a successful practice," observe David J. Brailer, MD, of the University of Pennsylvania, and David B. Nash, MD, MBA, of the Veterans Administration Medical Center, Philadelphia, in their editorial. "For diligent workers, medical practice was unique among the professions for its near guarantee of success."

Even choosing a specialty today can be risky, Brailer and Nash add. "Magnetic resonance imaging, for example, by accurately visualizing coronary artery occlusions, could obviate catheterization skills learned in the current cardiology fellowship. . . . Technological vulnerability can also promote subspecialization and professional rivalry."

Graduate medical education itself is threatened because of aggressive federal health care cost-containment policies, declining federal funding for research and education, and competition for patients by teaching hospitals, the researchers say. "Teaching hospitals may force residents to work without salary, or even charge them tuition," they add.

New payment methods also pose risks. Practice style and treatment costs may be compared to an arbitrary standard, and physicians treating sicker patients may be accused of practicing outside the "acceptable" range.

"Young physicians will have more difficulty in getting started than did their predecessors," predicts a second report, from the AMA's Council on Long-Range Planning and Development. They also will be burdened with higher educational debts. Tuition in public medical schools has risen almost eightfold since 1960, more than twice as fast as inflation, from $498 to $3959. Many physicians will opt for salaried positions, and may enter private practice much later, the report adds.

Other predictions: undergraduate medical (cont.)

*Researchers suggest conservative approach*

# Patients prefer more formality in doctors' dress and manners

A majority of patients believe doctors should wear white coats while on duty and find blue jeans inappropriate attire, reports a new survey. A substantial majority of those questioned also consider sneakers improper physician footwear. The survey results appear in the *Journal of the American Medical Association (JAMA).*

Along with preferences on attire, the survey, by Jocelyn J. Dunn of the Harvard Medical School and colleagues, found patients prefer formality in the way they address doctors. However, a concurrent poll of house staff at the same two hospitals where the patient survey was taken indicated many "had habits that were less formal than a substantial portion of their patients preferred."

The researchers questioned 200 patients on the general medicine services at Harvard's Brigham and Women's Hospital in Boston and H. C. Moffitt

(continued on p 96)

## Road to success (continued)

education will become even more expensive; there will be increased pressure to limit the growth in physician supply; some teaching hospitals will reduce or eliminate residency programs; economic pressures will create frictions among physicians; and there will be increasing separation between those who make economic decisions and those who make clinical decisions.

"The status of the medical profession could decline, and the profession could become a trade," the report says. "Physicians are likely to lose their exclusive authority over clinical decisions. . . . However, these same pressures are likely to make the physician's role as patient advocate even more important in the future."

In a third report, by John J. Norcini, PhD, and colleagues, of the American Board of Internal Medicine, say that FMGs had lower scores on the ABIM's 1982 certifying examination and had lower ratings from residency program directors than did US medical school graduates. The types of residency training programs also differed. "United States citizen FMGs were very similar to alien FMGs," the researchers add, emphasizing that US students who choose to train abroad may face similar obstacles in obtaining residency positions and practicing in the United States.

The study found that FMGs who scored well on the ABIM examination had also done well on the Educational Commission for Foreign Medical Graduates examination and were rated highly by their residency program director. "These data indicate attending a better residency program and performing well while in it improves one's chances of becoming certified. . . . Overall, the data argue for a more defined and detailed strategy of selecting FMGs to train in this country."

## In Memoriam

### 1985

| | |
|---|---|
| *William Hampton Garnier, MD* | *November 20* |
| *Jesse Ray Waltrip, MD* | *November 30* |
| *Charles F. Obermann, MD* | *December 30* |

### 1986

| | |
|---|---|
| *Alexander Poston, MD* | *January 3* |
| *Francis M. Duffy, MD* | *February 5* |
| *Edward L. Leonard, MD* | *February 14* |
| *William C. Tisdal, MD* | *February 24* |
| *Donovan Dillon Mosher, MD* | *April 4* |
| *Fred D. Switzer, MD* | *May 10* |
| *John D. Jennings, MD* | *May 12* |
| *Phillip Wade Jones, MD* | *May 18* |
| *Herbert L. Owen, MD* | *May 28* |
| *Marianne Elsbeth Kosbab, MD* | *June 13* |
| *William W. Rucks, Jr., MD* | *June 27* |
| *Ralph A. Smith, MD* | *July 27* |
| *Howard D. Tuttle, MD* | *August 3* |
| *Welborn W. Sanger, MD* | *September 19* |
| *William Carl Ewell, MD* | *September 20* |
| *Marcella Steel, MD* | *October 1* |
| *Terry Dwight Leming, MD* | *October 13* |
| *William Pat Fite, Jr., MD* | *October 30* |
| *Samuel Jackson McDaniel, MD* | *November 2* |
| *Iron Hawthorne Nelson, MD* | *November 12* |
| *John Robert Walter Spencer, MD* | *December 4* |

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## Doctors' dress (continued from p 94)

Hospital at the University of California–San Francisco. Seventy-four house staff at the two hospitals also were surveyed by mail.

Patient responses to questions on attire varied and many believed less formal variations in physicians' style of dress were appropriate, the study reports. About one-third of the patients had no preference on most issues, it notes.

However, 65% of those surveyed agreed that doctors should wear white coats when seeing patients and 52% said they should not wear jeans. More than one-third said male doctors should wear ties and women doctors should wear skirts or dresses instead of slacks; 27% believed sneakers were inappropriate.

On doctor-patient etiquette, only 18% of patients preferred to have physicians address them by their surnames. However, 74% preferred to address the doctor by title and surname, and only 10% were comfortable with being on a first-name basis with their physician, the report says.

Of the house staff answering the mail survey, tendencies in attire generally were conservative, the study finds. However, 72% of respondents said they sometimes or never wore white coats when seeing patients, 15% of the male doctors sometimes or never wore ties, and 82% of the women doctors sometimes or never wore dresses or skirts instead of slacks. Nine percent of the doctors sometimes wore jeans, and 43% sometimes or always wore sneakers.

In terms of etiquette, 88% of the doctors said they always or usually addressed patients by surname, according to the report, and 65% preferred to be addressed by title and surname.

While acknowledging that their results might not apply in all patient care settings, the researchers say their study is significant in pointing out the variability in patient preferences within the patient populations studied.

"Because of this variability, physicians in training as well as senior physicians may wish to adjust their etiquette to meet the expectations of their more conservative patients," the researchers conclude. "The physicians may prefer to adopt conservative habits to avoid displeasing a substantial segment of the patient population."


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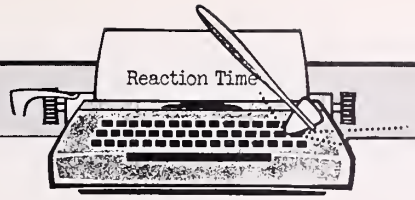
## Deaths

### Terry Dwight Leming, MD
### 1953 - 1986

Terry Dwight Leming, MD, a Sulphur native, died in his hometown October 13, 1986. A 1984 graduate of the University of Oklahoma College of Medicine, Dr Leming had returned to Sulphur in 1985 to begin his general practice.

### John Robert Walter Spencer, MD
### 1925 - 1986

Tulsa ophthalmologist Robert W. Spencer, MD, died December 4, 1986. Born in Akron, Ohio, Dr Spencer was graduated from the Vanderbilt University School of Medicine in Nashville, Tenn, in 1951 and established his practice in Tulsa in 1955. He was a member of the American College of Surgeons, the American Academy of Ophthalmology, and the Southern Medical Association.




## Specialists challenge trypsin use in hyaline membrane disease

*To the Editor:* In your October [1986] number, you carried a "Commentary" regarding the use of trypsin in neonatal respiratory distress syndrome by Dr J. William Finch. We congratulate Dr Finch for thinking and working for the betterment of neonatal care, but such reports should not appear in journals without review or editorial disclaimer.

This anecdotal and uncontrolled report, based on 34 previously unreported cases over more than twenty years, includes no data on infant size, gestational age, physical or radiographic definition of the "hyaline membrane disease," and no information about severity (blood gases, oxygen requirement, A-a gradients for oxygen or other therapy, duration of symptoms, progression to respiratory failure, and ventilation) either before or after the trypsin treatment.

The report gives no information about the population from which these patients were drawn except that it included 4095 babies. Dr Finch reports that "All of the patients responded. . . ." This must be a new record for a clinical trial — "100% of the time." Mild hyaline membrane disease (if that is what these babies had at all) is a self-limited process which clears as the child's lungs begin to secrete surfactant. These results, without placebo control, give *no* information about the effectiveness of the intervention.

Attempts have been made to dissolve the hyaline membranes in patients without success. Trypsin itself has not been tried, but the more specific approach of injecting plasmin to produce fribrinolysis has been studied with only minor improvements in HMD patients.[1] This leads to today's widely accepted view that the "hyaline membranes," those pink-staining layers within the alveoli on postmortem histologic slides, are "eosinophylic herrings" — markers of an *in vivo* layer of proteinaceous fluid which plays little or no role in the pathogenesis of the disease.

Even if proteolysis were helpful in this disease, one could hardly expect a high-molecular-weight enzyme like trypsin to reach the lung by mouth. If anything, the literature suggests that trypsin would be harmful to the baby with HMD since it is well established that trypsin *inhibitors* like alpha-1 antitrypsin are in short supply in the first day of life in HMD patients compared to controls[2] suggesting that trypsin *overactivity* may have a role in damaging

*(continued on p 98)*

## Muskogee *Daily Phoenix* runs Blair commentary on tort reform

Published as "Tort reform is on the state agenda" in the Muskogee *Daily Phoenix*, p 8B, Dec 24, 1986. Reprinted by permission.

Liability lawsuits are being filed by Oklahomans against Oklahomans at a record pace — the ninth highest lawsuit rate in all of the fifty states.

Businesses and professionals are the principal targets of this lawsuit binge — which isn't helping our sick economy any. The costs of goods and services go up, too, influenced by the rising costs of business and professional liability insurance. Some essential insurance coverages are not available now at any price. This causes the further retrenchment of our battered enterprises and professions which provide us with jobs and contribute greatly to the quality of our lives.

Instead of being Oklahomans pulling together for a better life, we're becoming plaintiffs and defendants, pulling apart.

"Oklahomans Against Lawsuit Abuse" is a coalition of statewide associations and organizations which represents over 300,000 citizens. According to recent polls, more than half of our population wants lawsuit abuse stopped.

In 1986, a tort reform bill we sponsored was passed in good shape by the House of Representatives, only to be bottled up and essentially ruined in the State Senate.

We will be back again in 1987 with another bill — our unfair laws which encourage too many lawsuits and excessive jury awards must be corrected in the best interests of society as a whole.

It is not right when one defendant among several co-defendants in the same lawsuit can be forced to pay the entire jury award even when the payor has minimal liability. Lawyers call this twisted justice the "deep pocket" approach.

*(continued on p 99)*

## Specialists challenge (continued from p 97)

the vasculature and producing the transudate known as a hyaline membrane.[3]

*Extreme* caution *must* be used in introducing new treatments for infants and children, surely the most vulnerable of our patients (remember Gantrisin, chloramphenicol, and even unlimited oxygen?). Where is the informed consent for the study of these patients? Federal and state regulations have been promulgated to protect patients' rights when investigations are conducted on patients who cannot consent for themselves.

The readership, especially those with less contact with the area of pediatrics and neonatology, should not be troubled by assertions of this sort in the pages of their JOURNAL. If Dr Finch wants us to believe his assertions, he must follow the rules of evidence for clinical trials *and* satisfy the referees at the JOURNAL that the method in fact works. Pediatric and/or neonatal review should be sought by the Editor-in-Chief before publication of material in this area of expertise.

Sincerely,

*Roger E. Sheldon, MD*
*Associate Professor of Pediatrics*
*Chief, Neonatal Section*
*OU College of Medicine*

*Mary Anne McCaffree, MD*
*Associate Professor of Pediatrics*

*Paul L. Toubas, MD*
*Associate Professor of Pediatrics*

*Pankaja S. Venkataraman, MD*
*Assistant Professor of Pediatrics*

*K. C. Sekar, MD*
*Assistant Professor of Pediatrics*

*Cody C. Arnold, MD*
*Assistant Professor of Pediatrics*

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3. Lieberman J, Evans HE: Hyaline membrane disease and antitrypsin deficiency. *Am Rev Resp Dis* 102:126-7, 1970.

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**A History of the National Library of Medicine. The Nation's Treasury of Medical Knowledge.** By Wyndham D. Miles. Washington: Government Printing Office, 1982. Pp 531, illustrated, $14.00.

Wyndham D. Miles, PhD, a member of the staff of the History of Medicine Division of the National Library of Medicine, has written an encyclopedic history of that unique institution. He traces the development of the library from the early nineteenth century through its growth as an international information center for biomedicine. It is well documented and contains a large number of interesting illustrations.

The book opens with an engaging preface by William B. Bean, MD. The library may be said to have originated when new US Army Surgeon General Lovell in 1818 needed a few books for his office. From that point the author traces the development of the library in an interesting and readable fashion. The library began as the "Library of the Surgeon General's Office." In 1840 a list of the library's tiny holdings was inscribed in a thin notebook. By the latter part of the Civil War a printed catalog noted 485 titles including 50 journals. The total number of volumes was a little over 2,000.

A great deal of the ultimate character of the present library can be attributed to the indefatigable efforts of John Shaw Billings, an army medical officer, who directed the library from 1865 to 1895. Under his direction the library produced its first general catalog and the *Index-Catalogue* and substantially expanded its holdings. Author Miles then traces the history from Billings's retirement to the present. He provides in detail a discussion of the accomplishments as well as the various crises and problems under various directors.

An important phase began in 1949 with the arrival of F. B. Rogers as the director. Two great achievements of this period were the development of MEDLARS and the move in 1962 from downtown Washington to the new National Library of Medicine building in Bethesda.

The passage of the Medical Library Assistance Act in 1964 permitted the library to expand its

*(continued)*

## Blair commentary (continued from p 97)

It is not right when a manufacturer of a product is indefinitely responsible for the product's safety, even though the product may have been modified by the user, or misused to the point where the injured party was in fact responsible for his own injury. A 10-year statute of limitations would be fair, and contributory negligence by the plaintiff should be taken into account.

It is not right when a defendant can be socked repeatedly for "punitive damages" throughout a stream of lawsuits brought by a series of plaintiffs for the same reason and based on the same evidence. "Punitive damages" are, in effect, a fine for especially irresponsible conduct. This class of damages is payable in addition to damages for medical bills, lost wages, pain and suffering and the like. One fine is enough; otherwise, double jeopardy is being unfairly imposed.

It is not right when the amounts of jury awards are calculated on the basis of exaggerated monetary estimates, but juries are deprived of knowledge about how much the plaintiff received for medical expenses and lost wages through private and public insurance programs. This double-recovery mischief game should be stopped!

It is not right that outrageous jury awards for so-called "noneconomic damages" are obtained without limitation for such intangible and unprovable complaints as "inconvenience," "pain," "emotional distress," and "humiliation." Such damage amounts frequently exceed the amounts awarded for actual economic losses which are objectively measurable. Other state legislatures have limited "noneconomic damages" in the range of $100,000 to $250,000. Oklahoma needs a lid on these frequently abused payouts.

These abuses of our civil laws will be addressed along with others in the bill to be introduced in January on behalf of our coalition.

If most of the state citizens favor a return to reason in our civil justice laws, as the polls show, surely the State Senate will respond through the enactment of meaningful tort reform in 1987.

*Don Blair*
*Executive Director*
*"Oklahomans Against Lawsuit Abuse"*

programs and to develop or acquire such activities as the Listen Hill Center for Research and Development, the National Medical Audiovisual Center, the Regional Medical Library Program, and information programs.

As Bean states in the preface about Miles, "He has made what might have been a mere chronicle into a story of imagination, of organizations, of ideas, and of many remarkably dedicated persons, military and civilian."

This is an excellent history of what has been termed "the central nervous system of American medical thought and research." The various chapters are enhanced by the informative notes at the end of each. However, the technical features of the book leave much to be desired. Published by the US Government Printing Office, the paper is of inferior quality and the binding is so poor that several pages in my copy became loose shortly after receipt of the book.

*Harris D. Riley, Jr., MD*
*Oklahoma City*

**Oklahoma Seminoles: Medicines, Magic, and Religion.** By James H. Howard in collaboration with Willie Lena. Norman: University of Oklahoma Press, 1984. Pp 251 with 81 plates, 27 figures, price not given.

At the outset of this review, I deem it proper to state that I think that the author has done an excellent job in arranging the material so that the reader can derive an optimum amount of both knowledge and enjoyment from the reading thereof.

In the preface it is explained how a smaller and more traditional segment of the Seminole tribe is still in Florida, while a larger segment is in Oklahoma, and this segment has altered more by means of adopting much of the white man's culture and modes of living. The book gives a cognitive review of the Oklahoma segment.

The author had a very close friendship with a man of the Oklahoma Seminole tribe, who was well versed in the culture and tribal customs of his people, a talented artist, and who had a sixth-grade education in the white man's schools. The author drew heavily upon the knowledge and talents of this man, Willie Lena, of Wewoka, Oklahoma. He also received assistance by having the plant specimens collected by Lena and himself identified (in terms of the white man's botany) by Ronald J. Zyrl and James McPherson of Oklahoma State University.

The book's foreword is an autobiographical sketch of this friend, whose great grandfather was Spanish, and was reared by his Seminole maternal grandparents and later in the home of his mother and his siblings, and he was thus familiar with both the Indians and white man's culture, was active in community affairs, and made and sold Indian trinkets.

**Some Seminoles fought with the Union and some with the Confederacy.**

The first chapter gives an extensive review of the tribe's history, relating the intertribal conflicts in Florida and Georgia, and how the white man from Spain, England, and the US used the Indian tribes for their advantage through trade, alliances, and by using the Indian as a buffer when needed to achieve his (the white man's) ends. The Creek and Seminole wars are reviewed, and the hardships the tribe suffered when it was forced to relocate in Oklahoma, in the 1835 "Trail of Tears," and at other times. In the Civil War, some Seminoles fought with the Union and some with the Confederacy. The discovery of oil on their land has helped them economically, some have been influenced by the Christian religion, some remained loyal to their old traditions, but most have accepted and adopted the culture of the larger society.

Through the remainder of this book we find discussions of superstitions, myths, witchcraft, magic, life-style, customs, and activities of a tribal people living close to nature as related by the author's authoritative Seminole friend, Willie Lena.

There are myths about the causes of their diseases, disorders, and symptoms, and we learn of their herbal and nonherbal remedies.

The author describes the ceremonialism of the

Seminoles, most of which takes place at a ceremonial or community center, also called "square ground," or "stomp ground." Beginning in April or May they have weekly ball games and monthly dances; their ceremonialism reaches its zenith in the Green Corn Ceremony in June or July, lasting through four days filled with Indian singing, dances, ball games, feasting, rituals, and commingling, with all wearing their best Indian attire. However, as time passes and their race merges more with the white man's culture, ceremonialism diminishes along with their enthusiasm.

In summary, the material in this book pertains to the Seminole tribe of American Indians in its turbulent march through more than four centuries, and tells how it has progressively become amalgamated with the white man's culture.

For one interested in this subject, this book is recommended as being both enjoyable and informative.

*Luke L. Ellenburg, Sr., MD*
*Greeneville, Tenn*

**Dictionary of Abbreviations in Medicine and Health Sciences.** By Harold K. Hughes. Lexington, Massachusetts: Lexington Books, D.C. Heath and Co., 1982. Pp 313, Price $23.00.

As stated in the preface, "Abbreviations are a way of life to most of us today, and we each learn those relatively few that are specific to our calling. . . . But in the health sciences, where there are technical vocabularies and millions of professionals, the large number of active abbreviations overtaxes our memories, and new ones appear in each journal issue. We are forced, therefore, to memorize those we use most often but to disregard the larger number we encounter without translation. It was to help the daily use of health-care abbreviations that this extensive list was compiled."

This dictionary covers usage in the United States, Canada, Great Britain, Ireland and other parts of English-speaking Europe, Australasia, southern Africa, and the United Nations. It contains more than 12,000 entries with some 20,000 meanings. The

*(continued)*

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health sciences are taken to include clinical, research, and production activities in all phases of professional care; food and energy resources; remedial education; veterinary science; and safety. In addition to the extensive dictionary content, the volume contains a guide to its use including some interesting, little known facts about the origin and other aspects of abbreviations, several tables explaining Greek letters, and various conversion tables.

The author, Harold K. Hughes, PhD, is a physicist who has pursued a 17-year career in business administration, followed by 5 years as vice president for academic affairs at a large university. He now teaches physics and mathematics at the State University of New York. He is the holder of many patents and the author of numerous publications in spectroscopy, standardization, creativity, and cybernetics.

Over a period of time I referred to this book when I encountered one or more of the myriad abbreviations used by medical students, house officers, and other physicians. Anyone who expects to find all of the abbreviations currently used in any one publication is quite naive. However, I did find many of them in this book.

In the course of this exercise, it was interesting to learn the number of different meanings that an abbreviation may carry for different individuals and the number of meanings that a particular abbreviation may have. For example, *CSF* to me always means "cerebrospinal fluid." However, it is also an acceptable abbreviation for "Canadian Schizophrenia Foundation," "colony-stimulating factor," "colored single female," and "cyanide-sensitive factor." As a matter of fact, there are very few abbreviations used in medicine which have only one meaning.

The appendix contains several helpful tables.

*Harris D. Riley, Jr., MD*
*Oklahoma City*

**The Ten Grandmothers: Epic of the Kiowas.** By Alice Merriott. Norman: University of Oklahoma Press, 1983, Tenth printing. Pp 289, 1 map, Indian art illustration at beginning of each chapter. Price not given.

A review of this book might well be analogous to viewing a broad and distant landscape by using two lenses with different focal lengths.

Through the first lens, the author, in a broad scope, reveals the generalities of the Kiowa Indian tribe, such as reviewing the sources for the material that related to this peace-loving, courteous, respected, nomadic, plainsman tribe of North American Indians — the Kiowas — who lived in the western part of Oklahoma; and in the introductory section, she explains the meaning of the word *Grandmother* as used in the title of the book. A Grandmother, she explains, was a collection of things considered sacred, wrapped in buffalo skin, and carefully entrusted to the care of a leading man in the tribe. Once each year these rolls were taken to a ceremonial tipi, unwrapped, and a tribal priest held a ritual over them, which supposedly gave them mystical and magical powers. After this ceremony they were rewrapped and stored for another year. Apparently, among the Kiowa tribe, there were ten such rolls, hence the title of the book.

**A *Grandmother* was a collection of things considered sacred.**

The Kiowas lived by hunting primarily buffalo, but also some deer. The meat of these animals, along with fruits from wild trees and plants, served as their food. The men hunted these animals. The women then cleaned the skins of meat and hair, tanned them, and used them to make clothing, moccasins, and their living quarters — the tipis. The women prepared the meals, attended to rearing the children, and maintained the tipi.

The Kiowas' family, community, and social life were mentioned. Each year they held a Sun Dance, which was a grand jubilee event where there were, in addition to general socializing, various contests, singing, dancing, and feasting.

The Kiowas had probably lived in this manner for hundreds of years, close to nature, which they looked to as their deity, and practiced brotherly love toward each other. They were self-sufficient and happy.

After leaving these general things about the Kiowas, the author then, as it were, switches to a telescopic lens and in thirty-one chapters gives a

close-range, detailed account of many facets of their tribal experiences.

All went well with them until the white man came to this continent and began to migrate northward, bringing his new and different customs, life-styles, etc, and imposing them upon the Indians, forcing them to undergo tremendous changes and adaptations, such as new living quarters (houses), clothing, religion (Christianity), missionaries, schools, foods, agriculture, grazing, and dividing the land into sections and fencing in these tracts. New methods in medicine were offered, mainly at Fort Sill.

Most of these innovations, although tolerated, were resented by the Kiowas, who preferred their previous mode of living, and reverted to it when chance permitted.

The book is divided into four sections, relating to the decrease and eventual disappearance of the grassy plains and buffalo, which slowly compelled the Kiowas to adopt the white man's way of life. Some Kiowas even served in the US armed services in our country's wars.

After reading this interesting book, one is left with a sort of after-image, much as one has after a flashbulb goes off when one's picture is taken. In this after-image, two quotations come to mind: (1) "The stand one takes on a controversial issue is largely determined by where fortune has ushered him to sit" —Anonymous, and (2) "He who complies against his will,/Is of the same opinion still" — Samuel Butler. Many questions come to mind as to which race was the better off, the Kiowas, or the usurping white race; and each side could give many reasons, both pro and con, to support their contentions and preferences.

Also, after reading the book, one might wonder (as I did) if the title of the book might easily have been something like this — *An Era of Transition, Foisted Upon the Kiowas*. Finally, it seems to be a fact that "Though the mills of God grind slowly,/they grind exceedingly fine" — F. Von Logau.

*Luke L. Ellenburg, Sr., MD*
*Greeneville, Tenn*

**Clinical Pediatric Dermatology.** By Sidney Hurwitz. Philadelphia: W. B. Saunders Co., 1981. Pp 481, illustrated, $75.00.

*Clinical Pediatric Dermatology* is an encyclopedic treatise of disorders of the integument in infancy and childhood. Containing 19 chapters, which include a listing of more than 400 different disorders, it is designed to provide the physician with information that will allow him to deal effectively with skin problems in the young.

The initial chapter gives an overview of the diagnosis of dermatologic problems. It includes a definition of terms and discusses certain basic principles such as regional distribution and morphologic patterns of various lesions. Following this is a discussion of general categories of cutaneous disorders such as skin disorders of the newborn, photosensitivity and photoreactions, disorders of the sebaceous and sweat glands, bacterial and protozoal infections of the skin, and others. The coverage of most disorders includes a discussion of histopathology of the appropriate lesion and each chapter is accompanied by an extensive list of references. The value of the book is enhanced by the large number of color photographs.

This is a comprehensive review of pediatric dermatology and represents an important reference. It will be of major use to all physicians who treat children.

*Harris D. Riley, Jr., MD*
*Oklahoma City*

***Haemophilus influenzae*. Epidemiology, Immunology, and Prevention of Disease.** Edited by Sarah H. Sell and Peter F. Wright, New York: Elsevier Biomedical, 1982. Pp 321, illustrated, $75.00.

In 1972 a conference was held at Vanderbilt University which dealt in depth with all aspects of *Haemophilus influenzae*. The proceedings of that conference were published (*Haemophilus influenzae*. Nashville: Vanderbilt University Press, 1973), and this publication has become a standard reference on this important subject. Since that time there have been substantial and significant efforts in investigation of the biology of this organism and the effects of infection with it. In 1981 a second conference, bringing together persons who have been engaged in *Haemophilus influenzae* research, was convened. This conference was sponsored by Vanderbilt University and the Tennessee Department of Public Health. Some fifty persons were present at the conference which was held in March 1981, and this book contains the proceedings.

This volume effectively updates the state of knowledge concerning *Haemophilus influenzae* and its infections. The volume is divided into five major

*(continued)*

## Book Shop (continued)

sections that characterize the major presentations and discussions, which are as follows: *Haemophilus influenzae* Disease and Epidemiology, Animal Models for *Haemophilus influenzae* Disease, *Haemophilus influenzae* Meningitis, Immunology of *Haemophilus influenzae* (Host, Bacterium), and Prevention. The latter topic or section is divided into studies dealing with immunization and those concerned with chemoprophylaxis.

This is an exellent book that provides important information concerning this significant topic. It should be available to the many different groups of investigators and clinicians concerned with the serious infections due to this organism. The editors, two physicians who have made important contributions to infectious diseases including those due to *Haemophilus influenzae,* have done a skillful job in synthesizing the vast amount of information presented.

*Harris D. Riley, Jr., MD*
*Oklahoma City*

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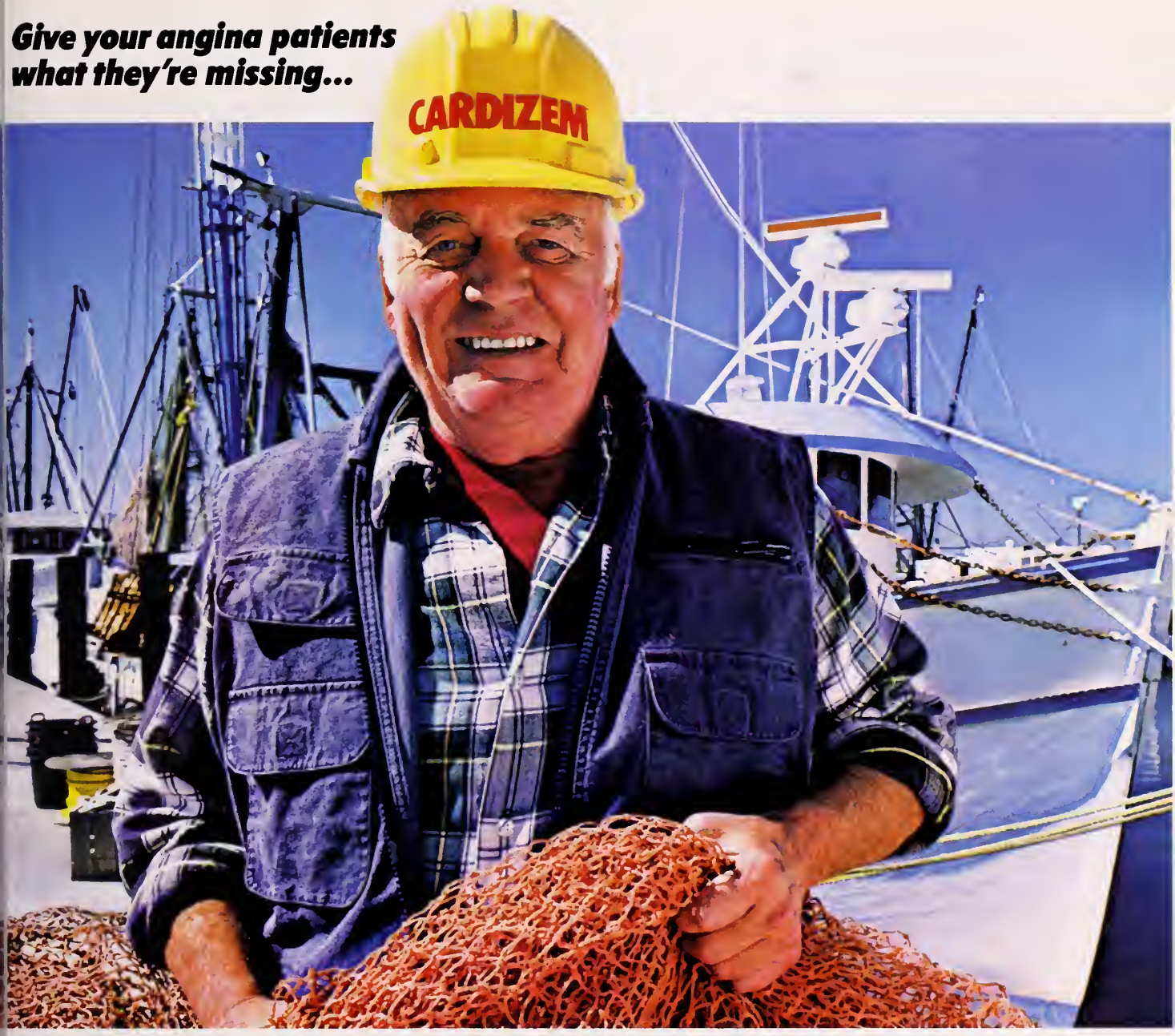


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*Please see brief summary of prescribing information on the next page.*

# CARDIZEM® FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

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**Brief Summary**
Professional Use Information

**CARDIZEM®**
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**CONTRAINDICATIONS**
CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

**WARNINGS**

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)

**PRECAUTIONS**
**General.** CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes, however, these changes were reversible with continued dosing.
**Drug Interaction.** Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)
Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.
**Carcinogenesis, Mutagenesis, Impairment of Fertility.** A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.
**Pregnancy.** Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.
There are no well-controlled studies in pregnant women, therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.
**Nursing Mothers.** Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.
**Pediatric Use.** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS**
Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.
In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.
The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%).

| | |
|---|---|
| Cardiovascular. | Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope. |
| Nervous System. | Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor. |
| Gastrointestinal. | Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase. |
| Dermatologic. | Petechiae, pruritus, photosensitivity, urticaria. |
| Other | Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties. |

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

Issued 7/86

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**References: 1.** Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers. Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983, 106(6):1341-1347. **2.** Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984,24(Dec):150-159. **3.** Johnston DL, Lesoway R, Human DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985;55:680-687. **4.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **5.** Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982;50(Sept):94-116.

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efore prescribing, see complete prescribing information in SK&F CO. :erature or *PDR*. The following is a brief summary.

**WARNING**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

**ontraindications:** Concomitant use with other potassium-sparing agents ich as spironolactone or amiloride. Further use in anuria, progressive nal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum itassium. Hypersensitivity to either component or other sulfonamide-rived drugs.

**'arnings: Do not use potassium supplements, dietary or otherwise, ıless hypokalemia develops or dietary intake of potassium is markedly ıpaired.** If supplementary potassium is needed, potassium tablets ıould not be used. Hyperkalemia can occur, and has been associated ıth cardiac irregularities. It is more likely in the severely ill, with urine ılume less than one liter/day, the elderly and diabetics with suspected confirmed renal insufficiency. Periodically, serum $K^+$ levels should be ıtermined. If hyperkalemia develops, substitute a thiazide alone, restrict intake. **Associated widened QRS complex or arrhythmia requires ompt additional therapy.** Thiazides cross the placental barrier and ıpear in cord blood. Use in pregnancy requires weighing anticipated ıefits against possible hazards, including fetal or neonatal jaundice, rombocytopenia, other adverse reactions seen in adults. Thiazides ıpear and triamterene may appear in breast milk. If their use is essential, e patient should stop nursing. Adequate information on use in children not available. Sensitivity reactions may occur in patients with or with-t a history of allergy or bronchial asthma. Possible exacerbation or :tivation of systemic lupus erythematosus has been reported with iazide diuretics.

**ecautions:** The bioavailability of the hydrochlorothiazide component of yazide' is about 50% of the bioavailability of the single entity. ıeoretically, a patient transferred from the single entities of triamterene ıd hydrochlorothiazide may show an increase in blood pressure or fluid tention. Similarly, it is also possible that the lesser hydrochlorothiazide ɔavailability could lead to increased serum potassium levels. However, tensive clinical experience with 'Dyazide' suggests that these conditions ve not been commonly observed in clinical practice. Angiotensin-nverting enzyme (ACE) inhibitors can elevate serum potassium; use th caution with 'Dyazide'. Do periodic serum electrolyte determinations articularly important in patients vomiting excessively or receiving renteral fluids, and during concurrent use with amphotericin B or rticosteroids or corticotropin [ACTH]). Periodic BUN and serum eatinine determinations should be made, especially in the elderly, abetics or those with suspected or confirmed renal insufficiency. ımulative effects of the drug may develop in patients with impaired renal nction. Thiazides should be used with caution in patients with impaired patic function. They can precipitate coma in patients with severe liver sease. Observe regularly for possible blood dyscrasias, liver damage, her idiosyncratic reactions. Blood dyscrasias have been reported in tients receiving triamterene, and leukopenia, thrombocytopenia, ranulocytosis, and aplastic and hemolytic anemia have been reported th thiazides. Thiazides may cause manifestation of latent diabetes ellitus. The effects of oral anticoagulants may be decreased when ed concurrently with hydrochlorothiazide; dosage adjustments may be cessary. Clinically insignificant reductions in arterial responsiveness norepinephrine have been reported. Thiazides have also been shown to crease the paralyzing effect of nondepolarizing muscle relaxants such tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic ood studies in cirrhotics with splenomegaly. Antihypertensive effects ay be enhanced in post-sympathectomy patients. Use cautiously in rgical patients. Triamterene has been found in renal stones in associa-n with the other usual calculus components. Therefore, 'Dyazide' ould be used with caution in patients with histories of stone formation. few occurrences of acute renal failure have been reported in patients 'Dyazide' when treated with indomethacin. Therefore, caution is vised in administering nonsteroidal anti-inflammatory agents with yazide'. The following may occur: transient elevated BUN or creatinine both, hyperglycemia and glycosuria (diabetic insulin requirements may altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), creasing alkali reserve with possible metabolic acidosis. 'Dyazide' :erferes with fluorescent measurement of quinidine. Hypokalemia is common with 'Dyazide', but should it develop, corrective measures ould be taken such as potassium supplementation or increased dietary :ake of potassium-rich foods. Corrective measures should be instituted utiously and serum potassium levels determined. Discontinue correc-e measures and 'Dyazide' should laboratory values reveal elevated rum potassium. Chloride deficit may occur as well as dilutional ponatremia. Concurrent use with chlorpropamide may increase the risk severe hyponatremia. Serum PBI levels may decrease without signs thyroid disturbance. Calcium excretion is decreased by thiazides. yazide' should be withdrawn before conducting tests for parathyroid nction. Thiazides may add to or potentiate the action of other anti-pertensive drugs. Diuretics reduce renal clearance of lithium and crease the risk of lithium toxicity.

**ıverse Reactions:** Muscle cramps, weakness, dizziness, headache, y mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other ermatological conditions; nausea and vomiting, diarrhea, constipation, her gastrointestinal disturbances; postural hypotension (may be ıgravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, ıresthesias, icterus, pancreatitis, xanthopsia and respiratory distress cluding pneumonitis and pulmonary edema, transient blurred vision, aladenitis, and vertigo have occurred with thiazides alone. Triamterene ıs been found in renal stones in association with other usual calculus ɔmponents. Rare incidents of acute interstitial nephritis have been ported. Impotence has been reported in a few patients on 'Dyazide', though a causal relationship has not been established.

**ıpplied: 'Dyazide' is supplied as a red and white capsule, in bottles of )00 capsules; Single Unit Packages (unit-dose) of 100 (intended for stitutional use only); in Patient-Pak™ unit-of-use bottles of 100.**

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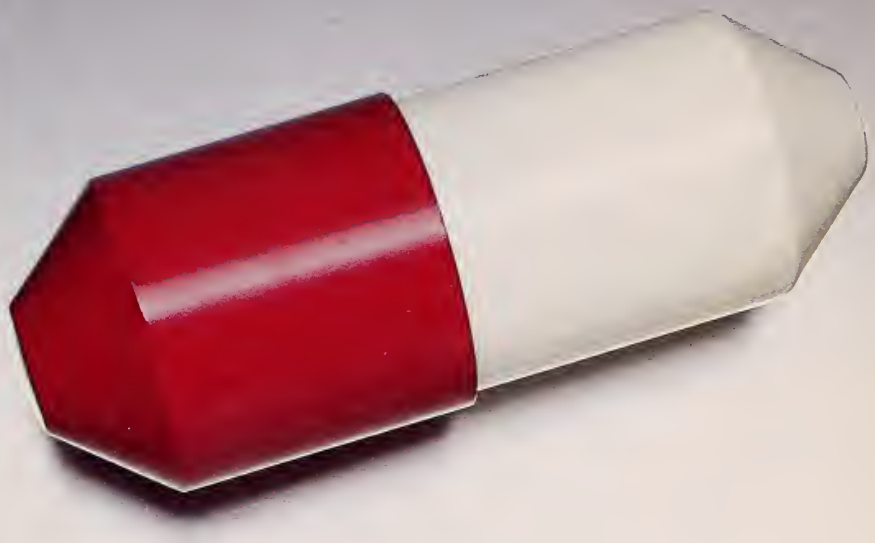

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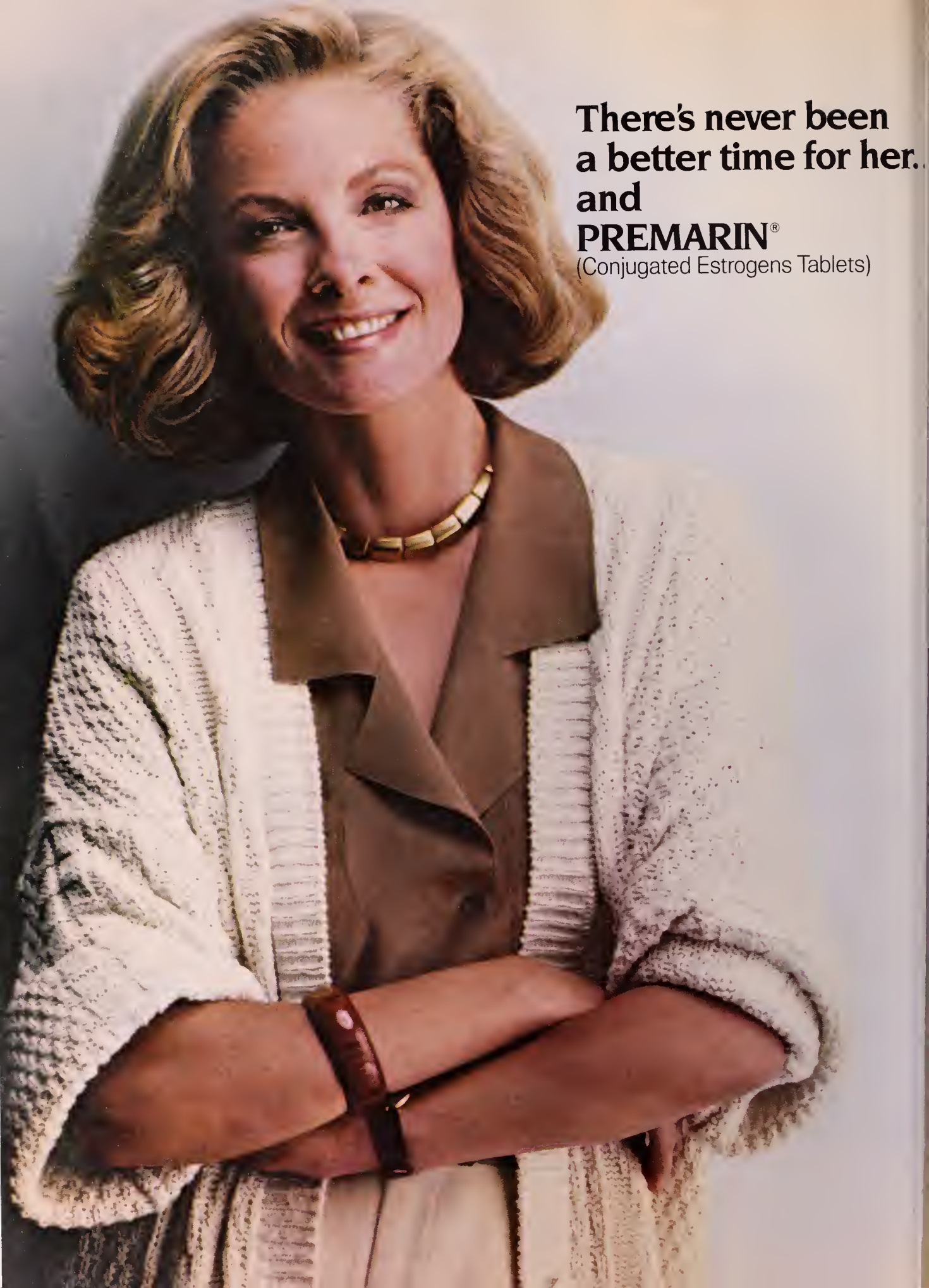
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and
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(Conjugated Estrogens Tablets)

# Now the evidence looks better than ever

## Significantly reduced risk of endometrial hyperplasia

Endometrial hyperplasia was significantly reduced when progestin was added to PREMARIN therapy for more than ten days a month.[1-4] The risk of endometrial hyperplasia may also be reduced through cyclic administration of unopposed, low-dose PREMARIN.

## Effect on lipids—an important feature

PREMARIN used alone does not adversely affect lipid levels. In fact, a clinical study has shown a significant increase in HDL cholesterol—from 49.7 mg/dL to 56.4 mg/dL—and decrease in LDL cholesterol—from 165.1 mg/dL to 138.1 mg/dL—after one year of therapy with PREMARIN, 0.625 mg.[5]

## Low-dose control of menopausal symptoms*

PREMARIN effectively relieves vasomotor symptoms, such as hot flashes. When estrogen deficiency is limited to atrophic vaginitis, PREMARIN® (conjugated estrogens) Vaginal Cream restores the vaginal environment to its premenopausal state.

## The most widely used, most extensively studied estrogen worldwide.

# PREMARIN®

(Conjugated Estrogens Tablets)

## Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

# For moderate-to-severe vasomotor symptoms

## PREMARIN®
(Conjugated Estrogens Tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

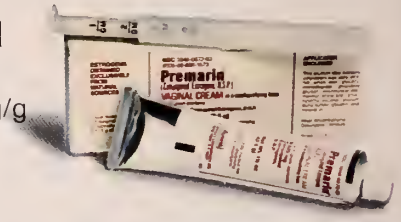
The appearance of these tablets is a trademark of Ayerst Laboratories.

# For atrophic vaginitis

## PREMARIN®
(Conjugated Estrogens)

Vaginal Cream

0.625mg/g




BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

**PREMARIN® Brand of conjugated estrogens tablets, USP**
**PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base**

1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA

Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy. Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses.

2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.

The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring. It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4.7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

**DESCRIPTION:** PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.

**INDICATIONS AND USAGE:** PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass).** Atrophic vaginitis. Kraurosis vulvae. Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae. PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

**Concomitant Progestin Use:** The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS.) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.

**CONTRAINDICATIONS:** Estrogens should not be used in women (or men) with any of the following conditions: 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease. 2. Known or suspected estrogen-dependent neoplasia. 3. Known or suspected pregnancy (See Boxed Warning). 4. Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

**WARNINGS:** Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning.) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility. There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease, or abnormal mammograms. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement, if has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk.

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed. Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.

**PRECAUTIONS:** Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Preexisting uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention.
b. Increased prothrombin and factors VII, VIII, IX, and X, decreased antithrombin 3; increased norepinephrine-induced platelet aggregability.
c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T4 by column, or T4 by radioimmunoassay. Free T3 resin uptake is decreased, reflecting the elevated TBG; free T4 concentration is unaltered.
d. Impaired glucose tolerance.
e. Decreased pregnanediol excretion.
f. Reduced response to metyrapone test.
g. Reduced serum folate concentration.
h. Increased serum triglyceride and phospholipid concentration. As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.

**ADVERSE REACTIONS:** The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea; premenstrual-like syndrome; amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance; aggravation of porphyria; edema; changes in libido.

**ACUTE OVERDOSAGE:** May cause nausea, and withdrawal bleeding may occur in females.

**DOSAGE AND ADMINISTRATION:**

**PREMARIN® Brand of conjugated estrogens tablets, USP**

1. *Given cyclically for short-term use only.* For treatment of moderate to severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.

2. *Given cyclically:* Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control. Osteoporosis—0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off).

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

**PREMARIN® Brand of conjugated estrogens Vaginal Cream**

*Given cyclically for short-term use only.* For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

**References:**

**1.** Whitehead MI, Townsend PT, Pryse-Davies J, et al: Effects of estrogens and progestins on the biochemistry and morphology of the postmenopausal endometrium. *N Engl J Med* 1981;305:1599-1605. **2.** Paterson MEL, Wade-Evans T, Sturdee DW, et al: Endometrial disease after treatment with oestrogens and progestogens in the climacteric. *Br Med J* 1980;280:822-824. **3.** Magos AL, Brincat M, Studd JWW, et al: Amenorrhea and endometrial atrophy with continuous oral estrogen and progestogen therapy in postmenopausal women. *Obstet Gynecol* 1985;67:496-499. **4.** Whitehead MI, Lane G, Siddle N, et al: Avoidance of endometrial hyperstimulation in estrogen-treated postmenopausal women. *Semin Reprod Endocrinol* 1983;1:1:41-52. **5.** Barnes RB, Roy S, Lobo RA: Comparison of lipid and androgen levels after conjugated estrogen or depo-medroxyprogesterone acetate treatment in postmenopausal women. *Obstet Gynecol* 1985;66:216-219.

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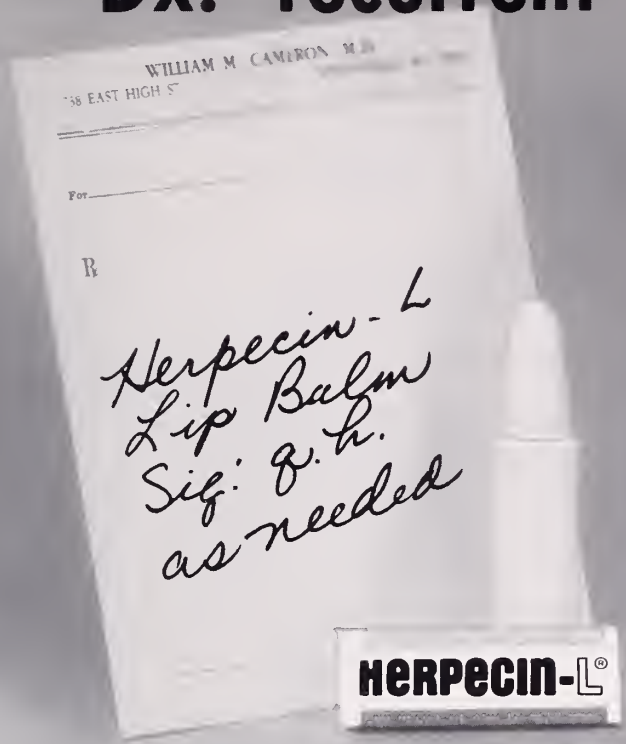
# Dx: recurrent herpes labialis

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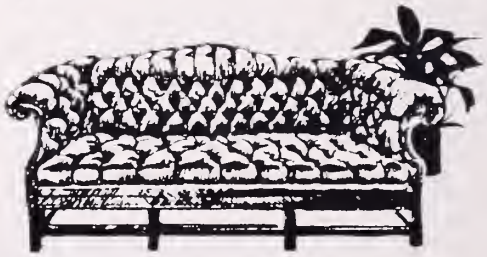

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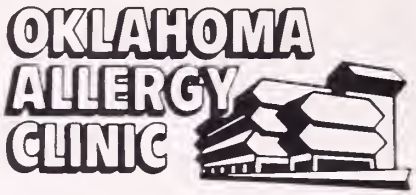


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Contemplating writing this article with Christmas rapidly approaching, my mind has been preoccupied with gifts and it occurred to me that health is one of the best gifts we have. Without good health, it's very difficult to enjoy all of the other wonderful aspects of life. Yet we are aware that many illnesses are related to lack of a healthy life-style. Auxilians have the potential and resources to promote a healthy life-style. As the State Health Projects Chairman, it is my hope that every auxilian will become involved in health education in his or her community.

There are two specific projects that I encourage all counties to support. The first of these is continued support of the MediFile project initiated last year. The State Medical office still has MediFile cards available. I urge all physicians to utilize these cards in treating their elderly patients. If you have questions regarding MediFile, or need more cards, just contact Beth at OSMA.

The other project that I encourage all counties to support is increasing public awareness of the teenage pregnancy problem in Oklahoma. A slide presentation relating to teen pregnancies is available through the State Auxiliary. It is appropriate for use at meetings of various organizations.

In addition to these two projects, I have asked each county health project chairman to target a particular need in their county. The National Auxiliary has a wealth of wonderful information available that can be utilized in health education. Publications are available on these topics: Child Abuse, Drinking and Driving, Drug Abuse, Food and Fitness, Community Services for Older Americans, Pre Natal and Post Natal Care, and Stress Management. There is also a comprehensive "Shape Up For Life" packet available that is excellent. There is no charge for the first copy of these materials to Auxiliary members.

What better gift can we give our community than to share our time and talents to help promote health education?

*Joy Quinn*
*State Health Projects Chairman*

■ **AMA officers and trustees would like to** make personal visits to group practices so they can describe what the AMA offers on behalf of physicians who practice in groups. If you would like to arrange for an AMA officer or trustee to visit your group practice, please call or write Dolores Pavela, Director, Office of Officer Services, AMA, 535 N. Dearborn, Chicago, IL 60610, (312) 645-4466.

■ **Farm families who consumed dairy products** tainted with the pesticide heptachlor had elevated blood levels of the chemical but exhibited no acute health effects from the exposure, researchers say in a letter to the *Journal of the American Medical Association (JAMA)*. Paul A. Stehr-Green, DrPH, of the Centers for Disease Control, Atlanta, and colleagues note, however, that their study of 13 families was not intended to address the long term of diseases like cancer. The study was conducted after pesticide residues were found in January 1986 in cattle feed sold in Arkansas, resulting in contamination of dairy products. Since farm families, at higher risk for exposure, showed no acute ill effects, the researchers say, "persons in the general community . . . are likely to be in no danger" from the contamination.

■ **Screening for drug and alcohol use and** risk factors is not routine practice in primary care pediatrics. But a study in the *American Journal of Diseases of Children* suggests a simple questionnaire completed in a doctor's office can gauge substance abuse risks in adolescents. Michael Klitzner, PhD, of the Pacific Institute for Research and Evaluation, Bethesda, Md, and colleagues administered the 42-item questionnaire to two groups, 97 youths from a drug abuse treatment program and 206 youngsters from a private pediatric practice. The questionnaire, which asked about various behaviors and the youths' relationship with their parents, was able to discriminate between the two samples, the study finds. It also appeared capable of discriminating alcohol and drug abuse within the samples, they say.

■ **Doctors working in the public sector are far** less likely to use the recently licensed *Haemophilus influenzae* type b (HiB) vaccine than private-sector physicians, a study in the *American Journal of Diseases of Children* says. The study, by Stephen L. Cochi, MD, of the Centers for Disease Control, Atlanta, and colleagues, surveyed 369 New Mexico physicians — pediatricians, family and general practitioners — providing primary care to children. Virtually all were aware of the vaccine, introduced in 1985, and it was in widespread use generally. However, while 69% of physicians in the private sector reported using the vaccine, only 11% of those in the public sector — like those at public clinics or in the Public Health Service — reported using it. This gap, the study says, "leaves a substantial proportion of children unprotected, since about one-half of all other vaccines routinely recommended for all children in the United States are given in public-sector vaccination programs."

■ **A study in the *Archives of Otolaryngology-Head and Neck Surgery*** reports excellent results in using a laser to treat allergic rhinitis (runny nose caused by house dust) that won't respond to traditional anti-allergy drugs and other therapy. Tomoshige Fukutake, MD, of the Kansai Medical University, Osaka, Japan, and colleagues, used a specially modified carbon dioxide laser to vaporize tiny vessels, causing scar tissue, inside the nose in the area where the allergic reaction is believed to occur. Of 140 patients, the study says, 131 showed improvement in symptoms one month after surgery, and 27 of 35 patients followed up more than one year postsurgery had excellent or good results. "This surgery can be done under surface anesthesia as an outpatient procedure, and it is painless, with no bleeding," the report says.

■ **A study in the *Journal of the American Medical Association (JAMA)*** outlines the rates at which a broad range of drugs cause skin rashes and similar allergic reactions in hospitalized patients. The study, by Michael Bigby, MD, of the Harvard Medical School, Boston, and colleagues in the Boston Collaborative Drug Surveillance Program, analyzes data on some 15,500 patients who received about 166,000 courses of drug therapy between 1975 and 1982. The study, an extension of earlier work by the same group, reports a 2.2% overall reaction rate. Drug-specific reaction rates were determined for 180 drugs or drug groups. Rashes were attributed to 51 drugs, with 75% of allergic skin reactions linked to antibiotics, blood products, and inhaled mucolytics (drugs that dissolve thick bronchial secretions). The antibiotic amoxicillin had the highest reaction rate, 51.4 reactions per 1,000 patients exposed.

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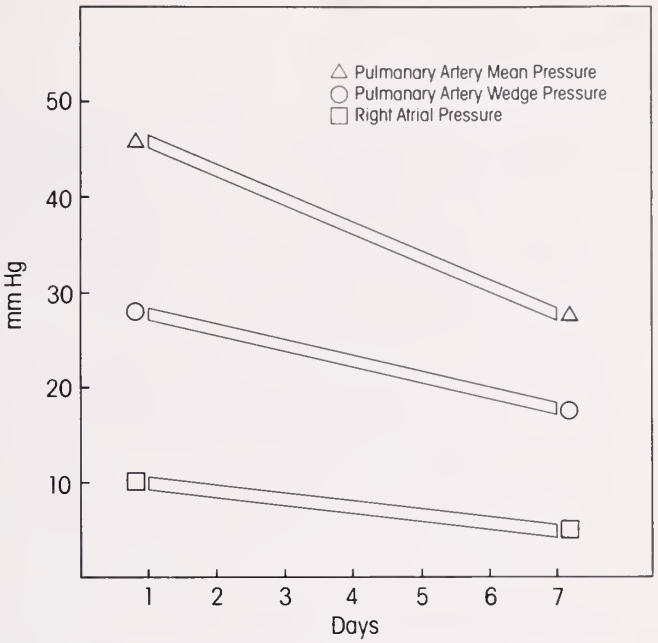


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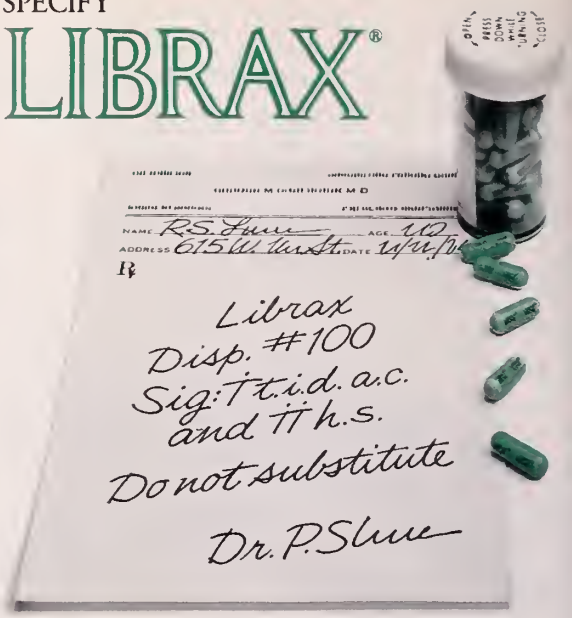
SPECIFY

# LIBRAX®




**Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide**

**Please consult complete prescribing information, a summary of which follows:**

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:
"Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.
Final classification of the less-than-effective indications requires further investigation.

**Contraindications:** Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
**Warnings:** Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.
*Usage in Pregnancy:* **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.**
As with all anticholinergics, inhibition of lactation may occur.
**Precautions:** In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
**Adverse Reactions:** No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

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# FLARE-UP

## SPASM AND PAIN CAN SIGNAL FUNCTIONAL GI DISORDERS*

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Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

ANTIANXIETY
ANTISECRETORY
ANTISPASMODIC

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of duodenal ulcer and the irritable bowel syndrome.

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Please see reverse side for complete product information.

# PHYSICIANS' RIGHTS:
## A DAY WITH THE JUDGES

## THE SIXTH OKLAHOMA LEGAL MEDICINE SEMINAR

**SPONSOR:** The American College of Legal Medicine

**DEDICATION:** In Memory of Judge Raymond Naifeh

**DATE & LOCATION:** Saturday, April 18, 1987
Raymond A. Young Conference Center
Baptist Medical Center
3330 Northwest Expressway
Oklahoma City, Oklahoma

**FACULTY:**
JOHN M. AMICK, Judge, District Court of Oklahoma County
CHRISTIAAN BARNARD, M.D., Ph.D., Scientist in Residence, Baptist Medical Center, OKC
W. EDWARD DALTON, M.D., Plastic Surgeon, OKC
DAVID RUSSELL, Judge, U.S. District Court, OKC
LEAMON FREEMAN, Judge, District Court of Oklahoma County
RICHARD M. GIBBS, M.A., M.D., J.D., LL.D., Boston (See below)
MARK R. JOHNSON, M.D., Internist, and Secretary, Oklahoma State Board of Medical Examiners, OKC
MARIAN P. OPALA, Justice, Supreme Court of Oklahoma
GARY W. RAHE, M.D., Hematologist, and Immediate Past President of the Oklahoma County Medical Society, OKC
WILLIAM R. SAIED, Judge, District Court of Oklahoma County
MARCUS B. SHOOK, M.D., Hematologist, and President of the Oklahoma Foundation for Peer Review, OKC
HARDY SUMMERS, Justice, Supreme Court of Oklahoma
ALMA WILSON, Justice, Supreme Court of Oklahoma
NAZIH ZUHDI, M.D., Director of Transplantation Institute, and Chief Heart Transplant Surgeon, Baptist Medical Center, OKC

**CHAIRPERSON:**
S. SANDY SANBAR, M.D., Ph.D., J.D., Cardiologist & Attorney, OKC

**CO-CHAIRPERSON:**
L. JEAN DUNEGAN, M.D., J.D., Surgeon & Attorney, Purcell

**LUNCHEON DISTINGUISHED GUEST SPEAKER**
RICHARD F. GIBBS, M.A., M.D., J.D., LL.D., FCLM
Past President, American College of Legal Medicine
Senior Anesthesiologist, Brigham and Women's Hospital
Assistant Professor of Anesthesia, Harvard Medical School
Vice Chairman of the Board of Directors of the Medical Malpractice
Joint Underwriting Association of Mass.

**SCOPE:**

The seminar will deal with current trends and issues pertaining to the RIGHTS OF PHYSICIANS. Actual cases involving physicians will be presented by the faculty doctors to panels of justices and judges who will address the physician's legal rights based on the facts and circumstances. The attenders will be provided with comprehensive looseleaf course materials. The subjects to be covered are listed below. Audience comments, questions and answers will be encouraged. The faculty is composed of excellent, highly knowledgeable and vastly experienced jurists, physicians and attorneys.

- - - - - - - - - - -

**PROGRAM**

| | |
|---|---|
| 9:30 a.m. | Registration, Coffee, Donuts & Exhibits |
| 9:50 a.m. | Welcome & Introductions |
| 10:00 a.m. | Rights Of Physicians By Statute & At Common Law |
| 10:30 a.m. | Right To Practice In A Hospital |
| 11:00 a.m. | Rights Under Federal Peer Review & Quality Assurance |
| 11:30 a.m. | Rights In Disciplinary Hearings By State Or Hospital |
| 12:00 noon | Luncheon |
| 12:30 p.m. | Distinguished Guest Speaks On Physicians' Credentialing & Discipling |
| 1:30 p.m. | Rights To Treat Or Refuse Treating A Patient |
| 2:00 p.m. | Rights In A Civil Lawsuit, State & Federal |
| 2:30 p.m. | Rights In Criminal Actions, State & Federal |
| 3:00 p.m. | Rights In Medical Malpractice Lawsuits |
| 3:30 p.m. | Refreshment Hour |

- - - - - - - - - -

**TUITION:** $75; For Residents, Interns & Students - $35

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8100 Glenwood, OKC, OK 73114

**CANCELLATION:** Refunds till April 12, 1987

**MATERIALS:** 200-Page Medical & Hospital Law Looseleaf Manual

**ACCREDITATION:** 5 hrs. Category 1, Continuing Medical Education

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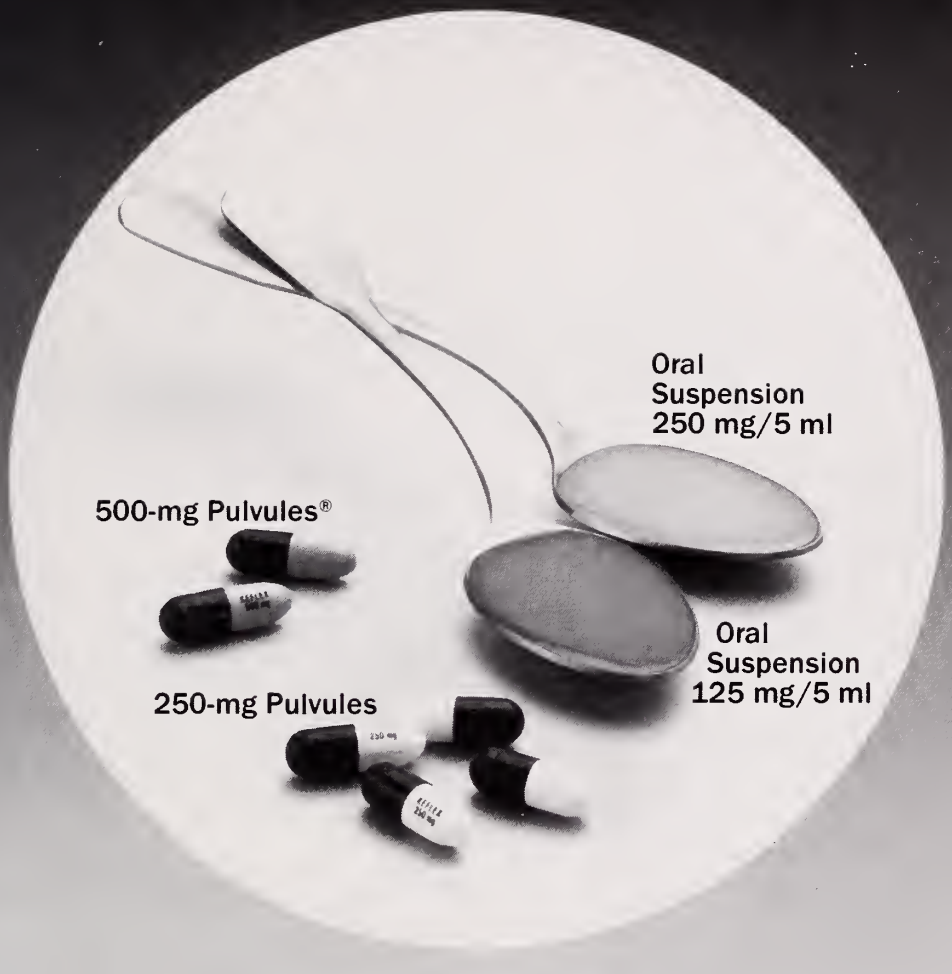


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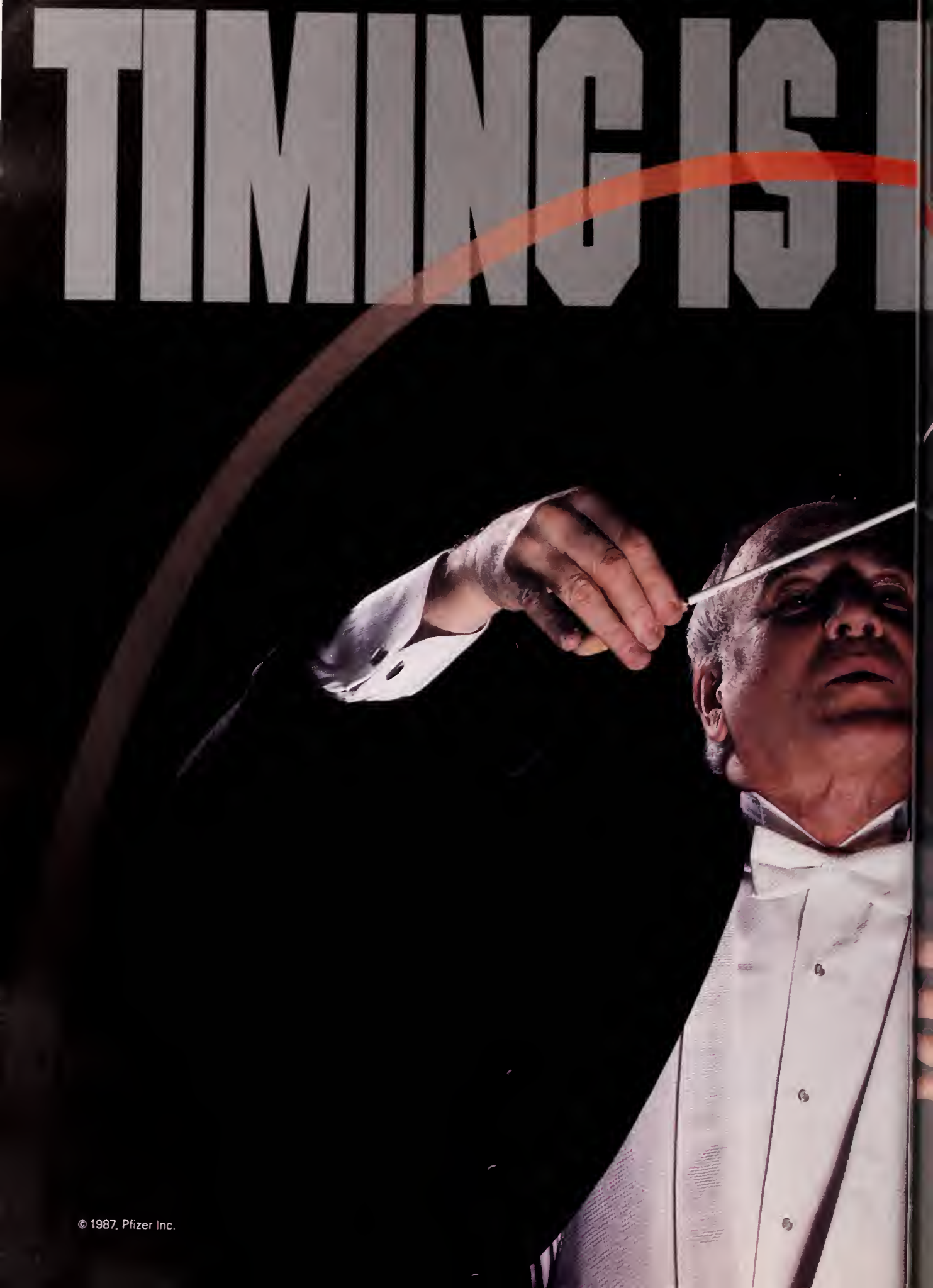


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# VERYTHING

## Effective control time and time again[1]

Effective control of fasting and postprandial glucose—patient after patient, meal after meal, year after year.

## Insulin when it's needed

Insulin levels are rapidly elevated in response to a meal, then return promptly to basal levels after the meal challenge subsides.

## Timed to minimize risks

Rapidly metabolized and excreted, with an excellent safety profile.[1] As with all sulfonylureas, hypoglycemia may occur.

**In concert with diet in non-insulin-dependent diabetes mellitus**

# Glucotrol®

(glipizide) 5-mg and 10-mg Scored Tablets

# SYNCHRONIZED SULFONYLUREA THERAPY

*Please see brief summary of Glucotrol® (glipizide) prescribing information on next page.*

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

**Reference:**
1. Sachs R, Frank M, Fishman SK. Overview of clinical experience with glipizide. In *Glipizide. A Worldwide Review*. Princeton, NJ: Excerpta Medica, 1984, pp 163-172

**GLUCOTROL® (glipizide) Tablets**

**Brief Summary of Prescribing Information**

**INDICATIONS AND USAGE:** GLUCOTROL is indicated as an adjunct to diet for the control of hyperglycemia in patients with non-insulin-dependent diabetes mellitus (NIDDM, type II) after an adequate trial of dietary therapy has proved unsatisfactory.

**CONTRAINDICATIONS:** GLUCOTROL is contraindicated in patients with known hypersensitivity to the drug or with diabetic ketoacidosis, with or without coma, which should be treated with insulin.

**SPECIAL WARNING ON INCREASED RISK OF CARDIOVASCULAR MORTALITY: The administration of oral hypoglycemic drugs has been reported to be associated with increased cardiovascular mortality as compared to treatment with diet alone or diet plus insulin. This warning is based on the study conducted by the University Group Diabetes Program (UGDP), a long-term prospective clinical trial designed to evaluate the effectiveness of glucose-lowering drugs in preventing or delaying vascular complications in patients with non-insulin-dependent diabetes. The study involved 823 patients who were randomly assigned to one of four treatment groups (*Diabetes*, 19, supp. 2:747-830, 1970).**

**UGDP reported that patients treated for 5 to 8 years with diet plus a fixed dose of tolbutamide (1.5 grams per day) had a rate of cardiovascular mortality approximately 2-1/2 times that of patients treated with diet alone. A significant increase in total mortality was not observed, but the use of tolbutamide was discontinued based on the increase in cardiovascular mortality, thus limiting the opportunity for the study to show an increase in overall mortality. Despite controversy regarding the interpretation of these results, the findings of the UGDP study provide an adequate basis for this warning. The patient should be informed of the potential risks and advantages of GLUCOTROL and of alternative modes of therapy.**

**Although only one drug in the sulfonylurea class (tolbutamide) was included in this study, it is prudent from a safety standpoint to consider that this warning may also apply to other oral hypoglycemic drugs in this class, in view of their close similarities in mode of action and chemical structure.**

**PRECAUTIONS: Renal and Hepatic Disease:** The metabolism and excretion of GLUCOTROL may be slowed in patients with impaired renal and/or hepatic function. Hypoglycemia may be prolonged in such patients should it occur.

**Hypoglycemia:** All sulfonylureas are capable of producing severe hypoglycemia. Proper patient selection, dosage, and instructions are important to avoid hypoglycemia. Renal or hepatic insufficiency may increase the risk of hypoglycemic reactions. Elderly, debilitated or malnourished patients and those with adrenal or pituitary insufficiency are particularly susceptible to the hypoglycemic action of glucose-lowering drugs. Hypoglycemia may be difficult to recognize in the elderly or people taking beta-adrenergic blocking drugs. Hypoglycemia is more likely to occur when caloric intake is deficient, after severe or prolonged exercise, when alcohol is ingested, or when more than one glucose-lowering drug is used.

**Loss of Control of Blood Glucose:** A loss of control may occur in diabetic patients exposed to stress such as fever, trauma, infection or surgery. It may then be necessary to discontinue GLUCOTROL and administer insulin.

**Laboratory Tests:** Blood and urine glucose should be monitored periodically. Measurement of glycosylated hemoglobin may be useful.

**Information for Patients:** Patients should be informed of the potential risks and advantages of GLUCOTROL, of alternative modes of therapy, as well as the importance of adhering to dietary instructions, of a regular exercise program, and of regular testing of urine and/or blood glucose. The risks of hypoglycemia, its symptoms and treatment, and conditions that predispose to its development should be explained to patients and responsible family members. Primary and secondary failure should also be explained.

**Drug Interactions:** The hypoglycemic action of sulfonylureas may be potentiated by certain drugs including nonsteroidal anti-inflammatory agents and other drugs that are highly protein bound, salicylates, sulfonamides, chloramphenicol, probenecid, coumarins, monoamine oxidase inhibitors, and beta adrenergic blocking agents. *In vitro* studies indicate that GLUCOTROL binds differently than tolbutamide and does not interact with salicylate or dicumarol. However, caution must be exercised in extrapolating these findings to a clinical situation. Certain drugs tend to produce hyperglycemia and may lead to loss of control, including the thiazides and other diuretics, corticosteroids, phenothiazines, thyroid products, estrogens, oral contraceptives, phenytoin, nicotinic acid, sympathomimetics, calcium channel blocking drugs, and isoniazid. A potential interaction between oral miconazole and oral hypoglycemic agents leading to severe hypoglycemia has been reported. Whether this interaction also occurs with the intravenous, topical, or vaginal preparations of miconazole is not known.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** A 20-month study in rats and an 18-month study in mice at doses up to 75 times the maximum human dose revealed no evidence of drug-related carcinogenicity. Bacterial and *in vivo* mutagenicity tests were uniformly negative. Studies in rats of both sexes at doses up to 75 times the human dose showed no effects on fertility.

**Pregnancy:** Pregnancy Category C: GLUCOTROL (glipizide) was found to be mildly fetotoxic in rat reproductive studies at all dose levels (5-50 mg/kg). This fetotoxicity has been similarly noted with other sulfonylureas, such as tolbutamide and tolazamide. The effect is perinatal and believed to be directly related to the pharmacologic (hypoglycemic) action of GLUCOTROL. In studies in rats and rabbits no teratogenic effects were found. There are no adequate and well controlled studies in pregnant women. GLUCOTROL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Because recent information suggests that abnormal blood glucose levels during pregnancy are associated with a higher incidence of congenital abnormalities, many experts recommend that insulin be used during pregnancy to maintain blood glucose levels as close to normal as possible.

**Nonteratogenic Effects:** Prolonged severe hypoglycemia has been reported in neonates born to mothers who were receiving a sulfonylurea drug at the time of delivery. This has been reported more frequently with the use of agents with prolonged half-lives. GLUCOTROL should be discontinued at least one month before the expected delivery date.

**Nursing Mothers:** Since some sulfonylurea drugs are known to be excreted in human milk, insulin therapy should be considered if nursing is to be continued.

**Pediatric Use:** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS:** In controlled studies, the frequency of serious adverse reactions reported was very low. Of 702 patients, 11.8% reported adverse reactions and in only 1.5% was GLUCOTROL discontinued.

**Hypoglycemia:** See PRECAUTIONS and OVERDOSAGE sections.

**Gastrointestinal:** Gastrointestinal disturbances, the most common, were reported with the following approximate incidence: nausea and diarrhea, one in 70; constipation and gastralgia, one in 100. They appear to be dose-related and may disappear on division or reduction of dosage. Cholestatic jaundice may occur rarely with sulfonylureas. GLUCOTROL should be discontinued if this occurs.

**Dermatologic:** Allergic skin reactions including erythema, morbiliform or maculopapular eruptions, urticaria, pruritus, and eczema have been reported in about one in 70 patients. These may be transient and may disappear despite continued use of GLUCOTROL; if skin reactions persist, the drug should be discontinued. Porphyria cutanea tarda and photosensitivity reactions have been reported with sulfonylureas.

**Hematologic:** Leukopenia, agranulocytosis, thrombocytopenia, hemolytic anemia, aplastic anemia, and pancytopenia have been reported with sulfonylureas.

**Metabolic:** Hepatic porphyria and disulfiram-like alcohol reactions have been reported with sulfonylureas. Clinical experience to date has shown that GLUCOTROL has an extremely low incidence of disulfiram-like reactions.

**Endocrine Reactions:** Cases of hyponatremia and the syndrome of inappropriate antidiuretic hormone (SIADH) secretion have been reported with this and other sulfonylureas.

**Miscellaneous:** Dizziness, drowsiness, and headache have been reported in about one in fifty patients treated with GLUCOTROL. They are usually transient and seldom require discontinuance of therapy.

**OVERDOSAGE:** Overdosage of sulfonylureas including GLUCOTROL can produce hypoglycemia. If hypoglycemic coma is diagnosed or suspected, the patient should be given a rapid intravenous injection of concentrated (50%) glucose solution. This should be followed by a continuous infusion of a more dilute (10%) glucose solution at a rate that will maintain the blood glucose at a level above 100 mg/dL. Patients should be closely monitored for a minimum of 24 to 48 hours since hypoglycemia may recur after apparent clinical recovery. Clearance of GLUCOTROL from plasma would be prolonged in persons with liver disease. Because of the extensive protein binding of GLUCOTROL (glipizide), dialysis is unlikely to be of benefit.

**DOSAGE AND ADMINISTRATION:** There is no fixed dosage regimen for the management of diabetes mellitus with GLUCOTROL; in general, it should be given approximately 30 minutes before a meal to achieve the greatest reduction in postprandial hyperglycemia.

**Initial Dose:** The recommended starting dose is 5 mg before breakfast. Geriatric patients or those with liver disease may be started on 2.5 mg. Dosage adjustments should ordinarily be in increments of 2.5-5 mg, as determined by blood glucose response. At least several days should elapse between titration steps.

**Maximum Dose:** The maximum recommended total daily dose is 40 mg.

**Maintenance:** Some patients may be effectively controlled on a once-a-day regimen, while others show better response with divided dosing. Total daily doses above 15 mg should ordinarily be divided.

**HOW SUPPLIED:** GLUCOTROL is available as white, dye-free, scored diamond-shaped tablets imprinted as follows: 5 mg tablet—Pfizer 411 (NDC 5 mg 0049-4110-66) Bottles of 100; 10 mg tablet—Pfizer 412 (NDC 10 mg 0049-4120-65) Bottles of 100.

**CAUTION:** Federal law prohibits dispensing without prescription.

**More detailed professional information available on request.**

ROERIG Pfizer A division of Pfizer Pharmaceuticals
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You would never let an oil field roughneck operate on your patient. As a physician, it makes even less sense for you to moonlight as an office manager.

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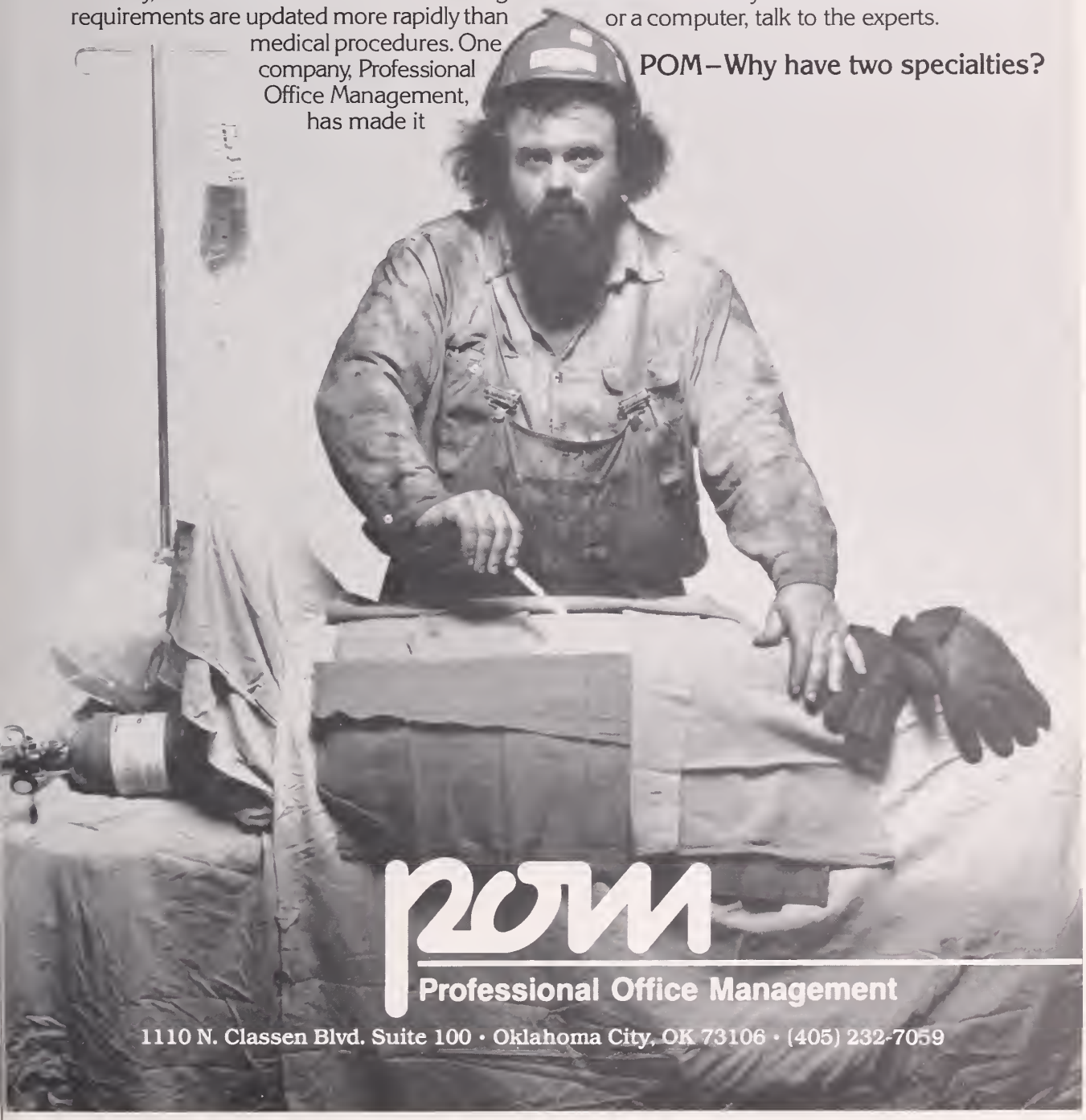

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# What You Pay For, You Get

Every time I read or hear an expression of outrage about overutilization of some Medicare reimbursed procedure, I am amused. Amused to tears.

When you pay to have something done but you pay nothing to determine the need to have it done, that something is going to be done. Simple fundamental fact. Fiscal insanity.

Almost two generations ago private health insurance companies began paying surgeons fixed fees for performing certain surgical procedures on their policyholders. They paid a reasonable amount of money for an appendectomy. They paid nothing for the determination that an appendectomy was not necessary.

Along came Medicare. It paid for almost anything done — but only in the hospital. Patients by the millions insisted they be put in the hospital. Soon there were not enough hospital beds. No problem. Taxes paid for more hospitals. Better equipment. More operating rooms.

Then, more doctors were needed to staff all the new hospitals. No problem. More medical schools were built and subsidized. Soon, more doctors were graduated. In their postgraduate training, they learned to do things they got paid for doing: to put patients in hospitals; to do endoscopic examinations, arteriograms, total parenteral feeding; they learned to implant pacemakers, bypass arteries, transplant hearts and kidneys and lungs and livers and corneas; they learned to maintain patients on respirators and dialysis; they learned chemotherapy, radiotherapy, isotope therapy, and laser therapy.

Not many doctors spent much time learning to do what they were not paid for doing: spending time with patients finding out what they needed to have done. Unfortunately, this function was given the title "cognitive services," and no one in the third-party-payor bureaucracy knew what the title meant. They were going broke paying for surgical services and hospital services, and whatever it was, they sure couldn't pay for cognitive services. What they needed was some way to decide if all those things that were being done to all those patients by all those doctors in all those hospitals really needed to be done.

In their majestic wisdom, the third parties developed a plan to replace the judgment of patient-selected, responsible, attending physicians with non–patient-selected, non-responsible, non-attending physicians. The judgments of these absent, anonymous physicians are referred to as DRGs when they are applied prior to the patient's admission to the hospital. When applied retrospectively, weeks or even months after the patient leaves the hospital, the substituted judgments are referred to as peer reviews.

All of this is necessary, the bureaucrats insist, because physicians are abusing the system. The truth is, the system has rewarded its abusers from its beginning, and it continues to do so. It still refuses to pay for the decision-making process essential to keeping patients out of hospitals and not submitted to much costlier, paid-for procedures. Instead, it is paying billions of dollars for last-minute pre-admission judgments and long-delayed, post-hospitalization, post-procedure judgments; paying those billions not to attending physicians but to a remote army of salaried, uninvolved bureaucrats, medical record librarians, nurses, clerks, lawyers, statisticians, computer operators, physicians, secretaries, writers, mail clerks, accountants, and political hacks.

Obviously those who are running this fiscal madness called Medicare do not understand a very simple truth: What you pay for, you get. Neither do they understand what they are earning; the enmity of every honest physician in this nation, the rapid degradation of our medical profession, and the eventual alienation of all Medicare patients as they begin to understand what it really is they're paying for and getting. *Instead* of medical care.

—*MRJ*

Dear Fellow Physicians:

As time passes, it is becoming more obvious that we in the medical profession are unable to unify to combat effectively the onerous changes being forced upon us by government and industry.

Greed, avariciousness, an occasional lack of understanding, and frequently, just plain fear of the unknown, appear to be preventing this unity which is essential for our protection. One remaining line of defense, however, has to do with maintaining the quality of our profession.

The problem arising is that the number of bright, intelligent students who wish to become physicians is decreasing. Eight to fifteen years of dedication, hard work, and personal sacrifice are needed to become capable of handling the intricacies of modern medicine. More and more, those really fine students who have the capabilities of handling these educational stresses are opting not to begin medical school.

This sounds tragic but is merely the law of supply and demand at work, particularly as the rewards of medicine diminish and the problems and difficulties of being a physician increase.

To allow this to continue is bound to create an inferior quality of medical care. This may be of no concern to our congressmen, society leaders, and industrial giants. Have you ever seen one of these groups opt for anything but "the best that money can buy" when they are ill?

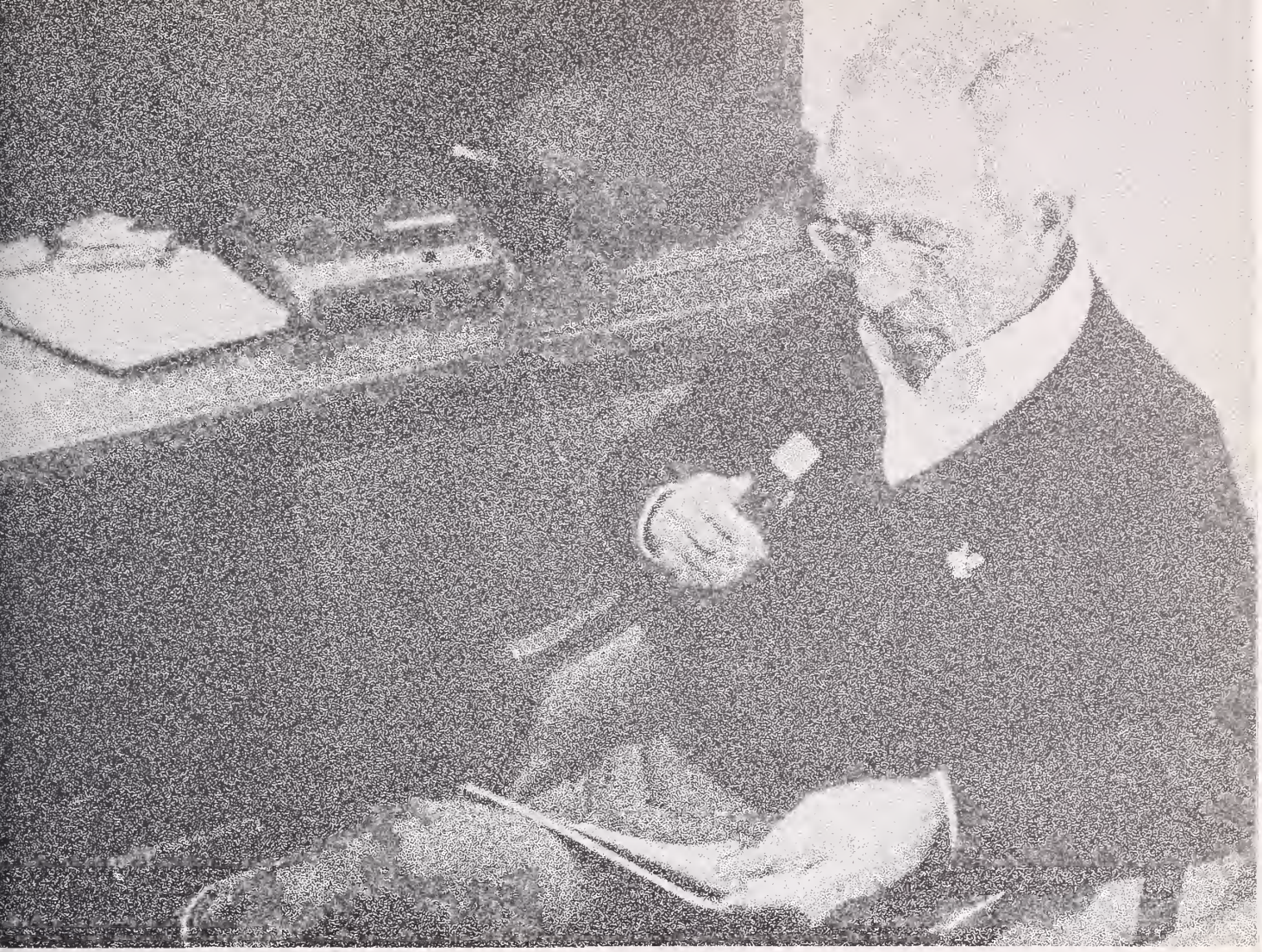
However, it is vital to those innumerable members of the public — the average Joe — who must be protected from inferior quality medical care, that the capability of the young people who will be providing this care in the future does not diminish.

Let us all join in unanimous support of the courageous stance of Doctor Donald Kassebaum, Dean of the OU College of Medicine, who has stood up to the pressures of our State Board of Regents and the legislators and firmly insists that only qualified and capable students be allowed to start a medical education.

Quality medical care is the right of all people — not just a privileged few — and those who do not meet acceptable standards must not be allowed to lower that level of quality that has been built up in this country at a considerable effort and expense to all.

It is vital that we do not back down nor allow students who simply do not have the powers to handle these difficult educational requirements to fill our school positions just to "fill a quota."

Norman L. Dunitz, MD



## Leaders in Medicine

# Richard E. Carpenter, MD

**Story by Richard Green**
**Photographs by J. Don Cook**

It is pure coincidence that Oklahoma's first neurologist also has turned out to be, by his colleagues' reckoning, one of the most respected and beloved. And it is this precise combination of intellect, creativity, assorted admirable personality traits, and of course, longevity that blend into Dr Richard E. Carpenter's unofficial title: the Father of Neurology in Oklahoma.

While many of his doctor friends and former neurology residents say the mantle fits, they know "Father of Oklahoma Neurology" sounds a bit too exalted and stodgy . . . too monumental for their friend. Dick Carpenter isn't on a pedestal. At 69 — remarkably young for an icon — he's still practicing neurology and leading University of Oklahoma neurology residents on hospital rounds. "I can't afford to quit; I'm still paying alimony," he says wryly.

Actually, he still loves the patient contact. Their stories ignite in him a need that is perhaps best characterized by neurologist-author Oliver Sacks: "Constantly my patients drive me to questions and constantly my questions drive me to patients." Those who have observed Dick Carpenter's practice in Oklahoma since 1949 have sensed these things as well as his compassion for many of his neurology patients who are, in a way, voyagers to unimaginable lands.

So two years ago, as a fitting way to honor their mentor and friend, a group of his former residents, led by OU neurologist Jeanne Ann King, decided to raise some money to create, in the neurology department at OU's Health Sciences Center campus, a library named for him.

Not coincidentally, the library's dedication was held in conjunction with the department's annual Tom Parker Memorial Lectureship, originated by Carpenter himself to honor the memory of his friend and neurology partner.

Dick cut the ribbon to open the Carpenter Neuroscience Library, which symbolized more than just the appreciation of his friends and colleagues. It also signified the beneficial influence he had been to hundreds of students and residents since he first started building his legacy of excellence in 1949.

* * *

His brother's appendicitis first stirred Dick Carpenter's interest in medicine. To a young schoolboy witnessing his parents' concern and brother's pain, the situation must have seemed serious if not grave. A surgeon from another town was called upon to operate. The object of all this attention must really be something, Dick thought. So he asked his parents for permission to watch the operation. They said no.

Rebuffed, Dick decided he would show them. If he became a doctor, he reasoned, he could witness any and all surgeries. Why, he might even perform

Dick Carpenter talks with colleague Richard Dotter as they prepare for another busy day.

the life-and-death surgeries himself. Despite this initial, highly questionable motive, he never considered any other line of work, even though his father, a geologist, would have liked Dick to follow in his professional footsteps.

When Dick was born in Bartlesville in 1917, his father, Everett Carpenter, was working for the oil company that soon became Cities Service. In fact, Everett Carpenter developed the company's geology department, reputedly the first in America. This investment soon paid huge dividends because he and the company's other geologists used their expertise to discover two enormous oil fields. When the news about the value of assessing surface geology got around, other oil companies were quick to hire staff geologists. Perhaps believing that more is better, Cities Service expanded its geology department, too, and before Everett Carpenter left the company, his department had about 125 geologists.

Everett met Neva Swan while they were both students at OU. He was studying geology under Charles M. Gould, who was married to Neva's sister. (OU's geology building is named for Gould.) Everett and Neva were married after graduation in 1911 and he took a job with the US Geological Survey. He studied and wrote comprehensive reports on water tables and on the prospects for agricultural development of arid regions in Utah and Nevada before returning to Oklahoma to get in on the ground floor of what was about to become the Big Oil Industry.

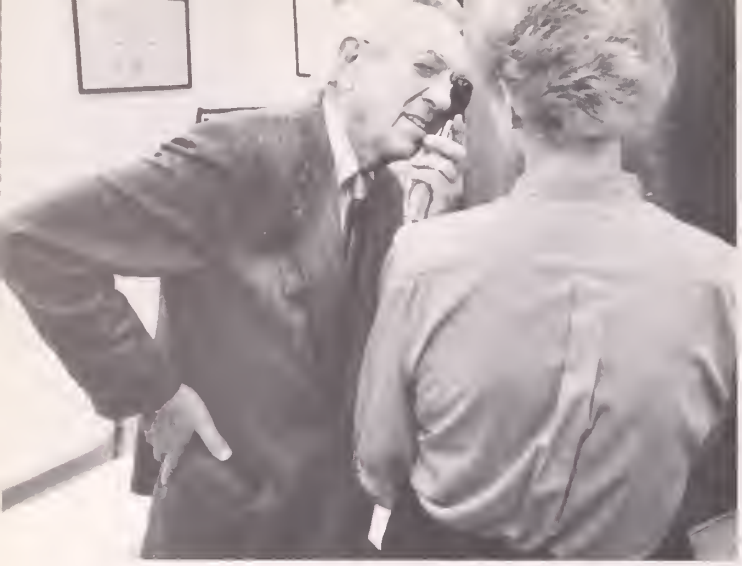
After Everett left Cities Service, he and some partners formed Emerald Oil Company and, using the same techniques that enriched shareholders for his former company, they proceeded over the next four years to drill 47 straight dry holes. "That's why we never had a lot of money in those days" Richard Carpenter remembers. "But Dad always had work, even during the Depression."

Richard's mother was involved in various social activities and served a term as president of the state Parents and Teachers Association. Richard was the second of the couple's three sons. Dan was two years older and Robert, two years younger. Because Everett was gone much of the time searching for the next big oil strike, Neva managed the family day to day. But Dick remembers that his father was the family's dominant figure. "My mother used to tell us that we were lucky to have such a good father. And we believed it."

Despite Everett's financial ups and downs, the family's equilibrium was unaffected until the boys' teenage years when Robert developed epilepsy, a debilitating and intractable illness that completely altered the course of his life. To a lesser extent, it also affected Dick's. "Robert's epilepsy probably guided me toward neurology."

The illness and its effects on Robert and the family undoubtedly started Dick's medical education early. And he would both suffer and profit from the experiences. He would see firsthand and quite personally that the patient's striving for identity under adverse circumstances can't be separated from the study of disease.

Robert had two kinds of seizures, the so-called petit mal, consisting of blank spells lasting from a



few moments to several hours, off and on for days, and the grand mal or convulsing variety. The seizures seemed to be cyclical, Dick remembers. "He'd go weeks and sometimes months without having any. Then, he'd have bunches of them."

To try to get Robert's seizures under control, his parents gave him a lot of attention and took him to both the Mayo Clinic and the Menninger Clinic. But in the days before antiseizure medicines, there wasn't much, if anything, that could be done for epileptics.

Still, Robert managed to graduate from Oklahoma State University with a degree in horticulture and then marry. But his seizures became more severe and frequent. He soon was unable to work at all.

Robert spent the balance of his life in an institution in Pauls Valley. "Yet, he wasn't bitter nor even particularly unhappy," Dick says. "He was able to tolerate his infirmity a lot better than I could have."

Five or six years before Robert's death in 1979, Dick started his brother on a new anti-seizure drug called Tegretol which allowed his last years to be seizure free.

* * *

The Carpenter family lived in Amarillo during most of the Great Depression. Though they didn't have much money, not much was required to live comfortably. "For 40 cents you could get into the dance and buy two Cokes," Carpenter remembers. And because Dick had several good friends there, his high school years were among the happiest of his life. In fact, he attended his fiftieth reunion in 1985.

He had had been an excellent student and wanted to attend either OU or the University of Texas. His parents ruled out OU to ensure that Dick wouldn't follow in the footsteps of his older brother Dan, who they felt paid more attention to his social life at OU than to his scholastic life.

His parents wanted him to attend Colorado University, and, being an obedient son, he enrolled rather unenthusiastically and completed his four-year complement in 1939. He remembers not having much fun; he had no transportation, and there were no nearby ski slopes anyway. Ice skating was popular but Dick had no talent for it. His grades and recommendations were so good, however, that on the suggestion of a biochemistry professor, he applied to and was accepted by one of the nation's best medical schools, the University of Chicago. For the 54 slots in the freshman class, there had been over 1,800 applications.

He intended to be a pathologist. "For two summers, I had worked for Dr Floyd Keller, who ran the Medical Arts Laboratory in Oklahoma City. I was just an unpaid 'gofer,' but I also learned how to do urine counts and blood tests, and that turned me on to pathology."

After his basic science years, Dick was awarded a year's fellowship in pathology, but what appeared to be a coveted step in the right direction actually proved to be a turning point. Since the fellowship was research oriented and the medical school had a reputation for independent study and thinkers, Dick decided to use his year to find the cause of rheumatoid arthritis.

In due course, he was floundering and then lost. Though he astutely decided that his career wouldn't be in medical research, the fellowship hadn't been a total loss. For example, during one of his clinical years, a man with a draining abdominal wound was admitted. "I knew from looking at the slide (of the tissue culture) that it was a certain type of fungus infection. I happened to tell this to a resident who at rounds presented the case as though he had made the diagnosis."

Also during that year, it dawned on Dick that people don't die from old age. "As part of the requirement, I did a lot of autopsies that year and there was an identifiable physical cause of death in every case," Dick says.

After his graduation in 1943, Dr Carpenter did two nine-month internships in Oakland, California. He signed on for the second nine months, as an assistant resident, simply because during the war

the hospital was in dire need of doctors. "There was practically no supervision, which was a shock at first."

The long working hours and sheer numbers of patients, however, gave him little time to reflect on many of his medical decisions. He didn't have much time to devote to his new family either.

Just before his medical school graduation, Dick had married a lab tech named Olga, and their first child, Michael, was born during the internship year. Over the next 15 years Dick and Olga had three more children, Susan, Molly, and Nancy.

* * *

Early during his internship, Dick decided he wouldn't be doing the surgeries he had envisioned as a youth. He was fascinated and stimulated by the art and science of diagnosis instead. So he took up an internal medicine residency at the specialty's unofficial "American Mecca," the Mayo Clinic. "It was quite a change for me because everything we did was closely supervised and scrutinized," says Dick. "And it was absolutely the best practice of medicine I had ever seen. When I have especially tough problems, I still refer (patients) to Mayo's."

While he was there, he got some bad news. "I had had a painful chronic back condition that caused me to be classified 4F by my draft board. Well, when my spinal area was x-rayed at Mayo's, they found some sclerosis in the sacroiliac region and figured I had rheumatoid spondylitis" (a crippling progressive inflammatory disease of the spine).

After absorbing the initial shock, Dick decided that since there was no treatment, he had little choice but to take it a day at a time. The pain wasn't constant and didn't worsen. As time went by, and it became apparent that the original diagnosis was incorrect, he was able to accommodate the discomfort.

His first hospital service at Mayo's happened to be in neurology. He enjoyed it so much that he eventually spent over half of his three-year internal medicine residency in neurology. He thought about switching residencies but figured that his training in neurology would help him, as a new internist, attract more referrals.

Dick accepted an offer to practice in nearby Duluth, Minnesota. He arrived in July and packed up and left the following spring. "I once heard someone say, 'The coldest winter I ever spent was one summer in Duluth,' and that about sums it up for me. It was time to head south."

* * *

Dick set up his practice in Oklahoma City in 1949. His parents and brothers were back in Oklahoma, and he wanted to be near OU's medical school. Through the county medical society, Carpenter announced he was subspecializing in neurology. Since there were no neurologists in central Oklahoma (and probably the entire state), sighs of relief among the medical community should have been almost audible.

Due to a severe shortage of hospital beds, however, staff physicians discouraged new members. "Saint Anthony wouldn't even give me an application," says Carpenter. He was granted privileges at Mercy and Wesley hospitals because he had "ins" at both places: Hugh Stout also had trained at Mayo's, and Floyd Keller had employed him at the Medical Arts Laboratory.

Dick joined OU's volunteer faculty that same year. He saw neurology patients at University Hospital one morning a week. At the time, neurology was a part of the psychiatry department, which was headed by Dr Coyne Campbell. Campbell, who also owned and operated a private psychiatric hospital (now called Willow View), paid Carpenter to do histories and physicals on patients admitted to his hospital. Moreover, Campbell began referring neurology patients to Carpenter.

His practice began to pick up as the Korean War heated up. At age 34, Carpenter was called to take a pre-induction physical exam. "I figured I'd rather be in the Air Force, so I joined up and was assigned to a 1,500-bed hospital at a basic training center in Geneva, New York. Two weeks later, I received notice from the Army that I was still 4F."

As the assistant chief of the medical service, Carpenter saw very few neurology patients during his two years of active duty. "It was the easiest two years I have ever had in medicine. The family was with me in Geneva. We joined a country club and I took up golf."

When Carpenter returned to Oklahoma City in 1953, he was still the only doctor practicing neurology willingly. Most neurology patients probably were being seen by neurosurgeons or psychiatrists.

Dick and his wife, Billie, derive a great deal of pleasure from the golf course that runs directly behind their Oklahoma City home.

Actually, it was probably self-interest that prompted two Saint Anthony neurosurgeons, Dr Harry Wilkins and Dr Jess Herrmann, to have him added to the hospital's staff.

As his availability and skills became known, Carpenter began attracting neurology referrals not only from the area but also from adjacent states. Still, he says he wasn't really comfortable practicing neurology for something like five to ten years. "I was seeing a lot of new things," he says. "But I knew I had been trained to do good neurological exams and felt like I performed them well. I could find the problem even if I couldn't always identify it."

His knowledge of the various dysfunctions and deficits — neurology's favorite words — was enriched by his experiences with his patients. Many of them had suffered strokes or had seizure disorders, movement disorders, or headaches. Many others had progressive degenerative afflictions, such as the dreadful Huntington's disease. And if he couldn't cure or even medically treat their illnesses, he learned by carefully and thoughtfully observing them how he could at least provide some comfort or reassurance.

He had joined the Oklahoma County Medical Society in 1949 and in 1960 became its president. He enjoyed the year as a learning experience and thought he did a reasonably good job, but was uncomfortable presiding over the meetings. He didn't like being the center of so much attention. And his one-year term at the top "satisfied whatever yen I had for a leadership role in organized medicine."

Furthermore, his practice was too busy to accommodate much outside activity. By then, Carpenter sometimes had a waiting list of up to six months. "It was appalling, really. I was making weekend rounds 50 weeks of the year. The pace was too hectic and I couldn't have kept it up for much longer."

Fortunately, the whirlwind ended in 1965 when he was joined by his first partner, Dr Tom Parker. "I met Tom while he was a medical student at OU and he was living next door to my brother," says Carpenter, who encouraged his interest in neurology.

After Parker completed an internal medicine residency, he studied neurology with Dr Gunter Haase at the University of Pennsylvania in Philadelphia. Haase recently had left OU to run a neurology residency program there, and Parker was one of his first residents.

When the young neurologist joined Carpenter, "it was a great relief. I could take every other weekend off," he says, smiling. Yet, in due course, Carpenter knew that the partnership was more than relief. "It was a blessing."

Despite their age difference, they became very close friends, and the qualities that they brought to their relationship enhanced the quality of each of their practices, Carpenter says. "Tom was energetic, delightful, and intellectually stimulating. And competitive. When he saw my patients on my weekend off, he delighted in making a diagnosis I had overlooked. And vice versa."

Just six years later, Parker developed acute stem cell leukemia. His doctors treated it and he battled it while continuing to see his patients. He was still seeing them a week before he died suddenly from a

brain hemorrhage in 1973. He was 41 years old. To memorialize him, Carpenter and others raised the money to start the annual Tom Parker Memorial Lectureship within OU's neurology department.

Though the fourteenth annual lectureship was held this past year, Carpenter says, "In a sense, you never stop grieving the loss of such a friendship."

Fortunately for Carpenter, two other neurologists, Richard Dotter and Marcelo Puiggari, had joined him before Parker's death. For by then, Carpenter had developed angina. His friend Dr C. Alton Brown says, "You could see sometimes he was in pain, but he never complained about it. He'd own up to it if I asked, but sometimes he'd just joke about it."

There didn't seem to be any alternative. Carpenter was told he was not a good candidate for by-pass surgery. "I could still do my job and play some golf and fish; I just couldn't run around the block very well." But as the 1970s progressed, so did the intensity and frequency of the heart pain.

Moreover, when the last of the Carpenter children left home, the glue that had held his marriage together dissolved. Dick and Olga separated in 1978 and were divorced two years later.

If 1980 was a low point in his life, the following year brought about a dramatic rebound. The impetus began when his doctors did another arteriogram and decided he had to have by-pass surgery. "Although I foolishly smoked cigarettes until the 1960s— and that didn't help things— we had a history of cardiovascular disease." His father died from a coronary and his mother died from a stroke, both at age 83, and his younger brother Robert, who had suffered so from epilepsy, died from a heart attack at 59.

Carpenter's surgery included seven by-passes. Like most doctors, he hated being the patient. He was in the hospital eight days but just a week later attended a medical meeting and in another two weeks resumed his practice.

His speedy recovery was in large part due to Billie Norvell. For years, she had been the head ICU nurse at Saint Anthony's and they had become friends. The friendship deepened after Carpenter's divorce and in 1981 they were married. He describes their marriage as something akin to an emotional rebirth for him. "We travel and play golf together and just have a wonderful time. If I could just get her to stop fishing with worms, then everything would be perfect."

The couple also has done a lot of traveling since their wedding five years ago, and plans even more extensive trips after Carpenter retires, "four or five years from now," he says. "I still like seeing my patients and working with the residents. It's stimulating and I think I'm still doing 'em some good. I know I'm doing myself some good."

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*J. Don Cook is a professional photographer currently on the staff of Oklahoma City's* Daily Oklahoman *newspaper. His work has taken him abroad on many occasions and has earned him a reputation as one of the state's finest photojournalists.*

## The Residents Write

# Legionnaires' Disease: A Historical Overview with Current Epidemiological and Clinical Perspectives

DAVID F. CORRAL, MD

**Legionnaires' disease — are you comfortable with your level of knowledge about this disease? Do you know when and how a patient presenting with signs of pneumonia should be worked up for Legionnaires' disease? Would you like a refresher on the major extrinsic risk factors which may predispose a patient to getting Legionnaires' disease, or reasonable preventive measures for patients particularly susceptible? Why not test your knowledge and experience against that of a recently graduated resident in medicine at our Health Sciences Center? If you disagree — let him know why.**

Test your knowledge: (Answers may be found at the end of the article)

1. Infectious spread of LD in humans has been *proven* to occur following:
   a. Direct contact with infected individuals
   b. Direct contact with contaminated fomites
   c. Inhalation of water vapor containing aerosolized bacteria
   d. Ingestion of bacteria with food or drinking water
   e. Two or more of the above
2. Which of the following increase the risk of acquiring LD?
   a. Smoking
   b. Alcoholism
   c. Immunosuppression
   d. Cancer
   e. All the above
3. Which of the following laboratory abnormalities is *not* likely to be associated with LD?
   a. Hyponatremia (less than 130 mEq/L)
   b. Leukocytosis between 20 and 30 K
   c. Hypophosphatemia (less than 2.5 mg/dl)
   d. Elevated LDH
   e. Elevated BUN
4. Which of the following measures has proven helpful as a preventive to the spread of LD bacteria in endemic areas?
   a. Hyperchlorination of water supplies to 5 ppm
   b. Increasing gastric pH to above 2.0
   c. Increasing the hot water temperature to 60° C
   d. Quarantine of infected individuals
   e. All of the above
5. Legionnaires' disease is commonly associated with all of the following except:
   a. Poor response to penicillins, aminoglycosides
   b. Nonproductive cough
   c. Patients who smoke
   d. Good response to antipyretics
   e. Temperature equal to or greater than 38.5° C

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Fig 1. Scanning electron micrograph showing Legionnaires' disease bacilli with tapered or bulbous ends. (Osmium-fixed, gold-palladium sputter coated Philadelphia 2, serogroup 1, from MHIH agar, 20,000 X.) Reproduced from Ref 14, p 136, by courtesy of Marcel Dekker, Inc.

Legionnaires' disease (LD), an unrecognized entity until its spectacular modern-day debut, instantly capitivated the attention of the medical community and the media. The well-publicized outbreak occurred in July 1976 during an American Legion convention at the Bellvue-Stratford Hotel in Philadelphia, Pennsylvania. Of 4500 Legionnaires attending the meetings, 182 contracted serious acute respiratory infections leading to 29 deaths.[1]

The cause of the illness puzzled investigators. No consistent bacteriologic species could be identified using standard staining or culture techniques. Finally, six months after the initial outbreak, a silver impregnation stain initially devised for staining rickettsiae led to the identification of significant numbers of intra-alveolar organisms.[1] These organisms (later named *Legionella pneumophila*) were ultimately found to be the cause of the outbreak.

Initial intensive efforts to culture the fastidious *L pneumophila* bacterium failed. Then, in a retrospective analysis of previously unclassified organisms, McDade and others at the Centers for Disease Control (CDC) found that an apparently identical organism had been described 30 years earlier. In 1947, a researcher named Elizabeth Jackson had isolated a previously unrecognized microbe (OLDA) from the blood of a laboratory worker with an acute febrile respiratory illness. Using culture techniques described by Jackson, the LD bacterium was successfully cultured.[2]

In 1977 the CDC revealed that a direct relationship between the OLDA organism and *L pneumophila* had been established based on DNA hybridization, immunofluorescence, serology, and growth properties.[2-4]

Patients infected with *L pneumophila* develop high serum antibody titers. The CDC was therefore able to conduct retrospective analyses of previously unsolved epidemics by examining patients' stored serums for *L pneumophila* antibodies.[5] Based on available sera, the CDC identified the earliest LD epidemic as having occurred in 1965 at Saint Elizabeth's Hospital in Washington, DC.[1] Seventeen epidemics involving 812 patients resulted in 112 deaths nationwide between 1966 and 1976 — all prior to the initial identification of the pathogen — were traced to legionella infections.[6]

## Current Estimated Incidence

Current estimates suggest that LD may account for as many as 25,000 pneumonias annually,[7] leaving a potential for thousands of deaths per year. Although clinical studies emphasize cases occurring in epidemics, sporadic infection may also be a common presentation of the disease. Prospective studies suggest that as many as one of every six or seven adults hospitalized with community-acquired pneumonia may have Legionnaires' disease (≅15%).[8,9] Nosocomial infection with *L pneumophila* is estimated to account for 3% to 12% of hospital-acquired pneumonias overall and up to 50% in hospitals with a previous history of LD outbreaks.[10,11]

## Microbiology

Legionnaires' disease is now recognized as being caused by a variety of organisms, all grouped within the family Legionnellaceae. The species *L pneumophila* has nine distinct serogroups,[12] with serogroup 1 accounting for up to 60% of all human infections.[11] Several other species are also recognized.

Microscopically the organisms appear as pleomorphic, nonacid fast, faintly stained gram-negative bacilli. Special silver impregnation stains (Dieterle, Gimenez) easily visualize the bacteria.[1,13] Electron micrograph studies (Fig 1) reveal an approximate size of 3 × 9 microns. Length varies and often is a reflection of growth conditions.[13,14]

LD bacteria will not grow on commonly used

bacteriologic media. Media containing amino acids, especially cysteine, are required to maintain growth of the fastidious organism.[13]

Legionella's uncanny ability to survive in the face of both the body's internal defenses and antibiotic therapy is explained by its protective cellular products. It typically produces beta-lactamases, which explains poor clinical responses observed following penicillin and cephalosporin therapy.[15] An important extracellular toxin inhibits in vivo killing. The toxin effectively impairs phagocytosis so that ingested organisms survive comfortably within macrophages, successfully escaping digestion. Only monocytes activated by *cytokines* have been shown to inhibit intracellular multiplication of *L pneumophila*. Inhibition occurs because monocyte phagocytosis is decreased and, consequently, less intracellular replication occurs. Cell-mediated immunity, *not* humoral immunity, is therefore the primary defense mechanism against this organism.[16]

Other toxins identified include weakly active endotoxin,[17] cytotoxin, and hemolytic exotoxins.[13] Some of the neurologic manifestations associated with Legionnaires' disease are also believed to be toxin-related.[14]

**Table 1. — Clinical Clues to Diagnosis of Legionnaires' Disease**

1. Pneumonic illness with multisystem abnormalities
2. Poor response to penicillins, cephalosporins, and aminoglycosides
3. High fever
4. Association with hospitalization or immunosuppression
5. Rapid progression of chest x-ray consolidation in patients with minimal sputum production

**Table 2. — Symptoms of Legionnaires' Disease***

| | Wadsworth Medical Center (61 patients) | Literature Review | Cumulative % |
|---|---|---|---|
| Cough | 92% | 85% (205/240)** | 87% |
| Chills | 77% | 72% (164/228) | 73% |
| Diarrhea | 47% | 33% (74/225) | 36% |
| Dyspnea | 36% | 62% (116/187) | 56% |
| Chest pain | 33% | 35% (13/37) | 34% |
| Headache | 28% | 48% (103/215) | 43% |
| Sweats | 26% | | |
| Nausea, vomiting | 25% | 23% (24/104) | 24% |
| Myalgia, arthralgia | 20% | 49% (105/214) | 43% |
| Abdominal pain | None | 15% (16/109) | |

* Reproduced from Kirby BD, Snyder KM, Meyer RD, Finegold SM: Legionnaires' Disease: Report of Sixty-Five Nosocomially Acquired Cases and Review of the Literature. *Medicine* 59(3):188-205, © by Williams and Wilkins, 1980.

** Number of patients with symptom/number of patients evaluated.

## Epidemiology

LD occurs worldwide, most commonly in association with aquatic ecosystems. The organism is rarely found in the soil unless it is fairly moist. Blue-green algae are commonly seen in association with legionella and are thought to provide an essential carbon source. Legionella can withstand a wide range of temperatures from 6°C to 67°C, accounting for their fairly widespread existence. The incidence of LD is highest from June to October, although it occurs year round. People in midwestern and northeastern states are more commonly affected. Person-to-person transmission has *not* been proven.[13,14]

Evidence consistently points to the fact that legionella infection results from the airborne spread of bacteria into the lung.[13] Investigations of multiple nosocomial outbreaks have helped elucidate important transmission modes and associated risk factors. In 1978 a Memphis hospital reported 39 cases of LD over a one-month period affecting housestaff, visitors, and employees. The infection was linked to a contaminated cooling tower which aerosolized water vapor into an air-conditioner intake. Once the water tower was removed, no further cases occurred.[18]

Twelve cases of LD were reported in renal transplant patients in a British transplant unit. Aerosolized bacteria originating from the showers in the unit were linked to the infection.[19] The longest continuing epidemic to date occurred from May 1977 to February 1982 at the Wadsworth VA hospital in Los Angeles, where over 218 confirmed cases were ultimately linked to a contaminated hospital water system.[20] Similarly, researchers recently confirmed the presence of LD bacteria from hot water fixtures in Chicago apartments, where the hot water temperatures were lower than 60°C.[21]

The possibility of acquiring LD by merely ingesting contaminated water has been questioned. Normally, gastric acidity provides a protective barrier to most bacterial infections. *L pneumophila* serogroup 1, however, has been shown to survive 30 minutes in HCl despite pH of 2.0. A pH of 1.7 is inhibitory following a one-minute exposure; however, the pH in many gastric tracts is not that low. To date, *no* human cases have been presented to support the theory that ingestion of organisms may lead to infection. Seroconversions and infections in *guinea pigs*, however, have been shown to occur experimen-

**Fig 2.** (Left) Admission chest radiograph showing minor left lower lobe consolidation. (Right) Progression in three days to complete consolidation of left lower lobe, with patchy infiltration within right upper lobe. Patient recovered completely. Reproduced from Ref 28, p 588, by courtesy of the American Medical Association and with consent of the author.

tally after LDB ingestion — especially with *elevated* gastric pHs.[22]

Hyperchlorination of tap water has been studied for its inhibitory effect. The standard concentration of chlorine in most tap water systems is 0.2 parts per million (ppm). The minimal chlorine concentration capable of inhibiting legionella growth is 0.65 ppm chlorine after one-hour exposure.[23] In some endemic areas, despite hyperchlorination of water systems to minimal inhibitory levels, LD infections have reoccurred.[24] Chlorination of water over 5.0 ppm has been shown to inhibit growth after one-minute exposure and may be necessary to effectively inhibit LD bacteria in endemic areas. Additionally, increasing hot water temperatures to above 70°C has been shown to be helpful.[21] UV light treatment of water may also be effective.[25]

## Clinical Features

Inhalation of legionella from an environmental source produces an acute alveolar infection, with a clinical response which ranges from asymptomatic seroconversion to death. In general, individuals of any age may be affected, although the current median age of clinical cases is 55 years, and less than 10% of cases have been in persons under 20 years of age.[14] There is a three-to-one male predominance without any racial predilection.[26]

The most important extrinsic risk factors include smoking (four-fold risk) and alcoholism (five-fold risk).[14] The major underlying predispositions include immunosuppression (nine-fold risk in one study),[10] cardiac disease, malignancy, rheumatological/inflammatory diseases, chronic obstructive lung disease, renal disease, and diabetes.[13]

Important tips that should make Legionnaires' suspect in the diagnosis are shown in Table 1.

The vast majority of patients with Legionnaires' disease develop pneumonia. Infection with *L pneumophila* is most commonly recognized as an atypical pneumonia that occurs frequently in immunocompromised patients.[13] A highly virulent strain of legionella has been described which causes a nonpneumonic form of Legionnaires' disease called Pontiac fever. This form of LD is highly associated with air conditioners although it is apparently an uncommon infection.[14]

The most common clinical history in patients with pneumonic Legionnaires' disease is that of an insidious, nonproductive cough which becomes productive over a week. Pleurisy, tachypnea, and moist rales are common. Fever correlates highly with LD, with over 90% of patients presenting with temperatures of 38.9°C or more. Temperature elevation may occur in steps or abruptly, is usually associated with a relative bradycardia, and is usually unaffected by antipyretics or steroids.[14] Gastrointestinal symptoms of watery diarrhea are present in up to 50% of the cases. Headache is a common neurological symptom, and 25% to 30% may have mental confusion, with or without hallucinations. Peripheral neuropathy and ataxia have been reported.[20]

## Complications

Up to 30% of patients with underlying disease may require an ICU admission for management of their pneumonia, and 15% to 20% of those may require intubation and mechanical ventilation. Structural pulmonary complications may progress to cavitation, empyema, and scarring.[27]

Patients with a previous history of renal insufficiency may progress to renal failure. Permanent neurologic dysfunction is a rarely reported complication. Less common complications include pericarditis with or without myocarditis, pleuritis, rhabdomyolysis, encephalitis, bronchospasm, and rash.[14,20]

## Laboratory Findings

Chest x-rays usually show unilateral patchy segmental alveolar infiltrates with the lower lobes most commonly affected. Spread to adjacent ipsilateral lobes may be rapid with extensive consolidation (Fig 2).[13] Small pleural effusions may be present; rarely, an empyema or pneumothorax occurs.[13,14,20]

A commonly found laboratory abnormality in the Wadsworth VA hospital study was leukocytosis with a left shift. Greater than ten thousand white blood cells/cu mm were seen in 78% of cases, with most counts falling between ten and twenty thousand/cu mm (Table 3).

Sputum gram stain typically shows scant to moderate numbers of polymorphonuclear leukocytes with an absence of any predominant flora.[1,13]

## Atypical Pneumonia Differential Diagnoses

Many infectious agents can produce disease mimicking legionella pneumonia, including *Mycoplasma pneumoniae, Chlamydia psittaci* (psittacosis), *Coxiella burnetii* (Q fever), *Rickettsia rickettsii* (Rocky Mountain spotted fever), salmonella spp (typhoidal syndrome), *Histoplasma capsulatum, Coccidioides immitis, Blastomyces dermatitidis,* influenza, and other viruses. In narrowing the differential, it is important to establish a thorough data base relating to recent travel, occupational exposure, and exposure to birds, arthropods, animals, and their by-products.[13,14]

## Diagnosis of Legionnaires' Disease

The best diagnostic approach combines clinical suspicion with rapid direct fluorescent antibody stains, and culture, followed by indirect fluorescent antibody titers in serum drawn acutely, and again after one month's convalescence. The most effective way to maximize sensitivity (over 70%) and specificity (approaching 100%) is to combine all three diagnostic methods.

Table 3. — Laboratory Findings in Legionnaires' Disease*

| | Wadsworth Medical Center | Literature Review |
|---|---|---|
| Leukocytosis (≤10,0000/mm²)** | 78% (45/58) (range, 10,100 to 36,400) | 45% (126/279) |
| Hyponatremia (130 to 150 mEq/L) | 54% (30/55) (range, 117 to 129) | 68% (17/25) |
| Hypophosphatemia (2.5 to 4.5 mg/dl) | 51% (22/43) (range, 1.2 to 2.4) | Not reported |
| Increased LDH (90 to 200 IU/L) | 88% (36/41) (range, 205 to 850) | 45% (5/11) |
| Increased SGOT (10 to 50 IU/L) | 49% (21/43) (range, 53 to 725) | 65% (13/20) |
| Increased alkaline phosphatase (30 to 85 IU/L) | 49% (20/41) (range, 96 to 805) | 62% (8/13) |
| Increased bilirubin (0.2 to 1.2 mg/dl) | 15% (7/47) (range, 1.4 to 4.5) | Data too limited |
| Proteinuria | 45% (15/38) (range, + to + + +) | 25% (26/103) |
| Hematuria | none | 16% (16/100) |
| Renal failure | none | 13% (18/134) |

* Reproduced from Kirby BD, Snyder KM, Meyer RD, Finegold SM: Legionnaires' Disease: Report of Sixty-Five Nosocomially Acquired Cases and Review of the Literature. *Medicine* 59(3):188-205, © by Williams and Wilkins, 1980.

** Normal value for WMC laboratory.

**Direct fluorescent antibody** (DFA) is the most rapid (in a few hours) method available for identifying *L pneumophila.* Clinical specimens (sputum, pleural fluid, etc) are stained by fluorescein-labeled antibodies to the organism, which allows microscopic identification. This method is most successful using sputum and is positive in about 50% of patients with LD. DFA sputum examination is very specific (95%), but only moderately sensitive (70%).[29] The test is available at larger and reference laboratories for about $20.

**Culture,** using a charcoal yeast extract and appropriate amino acid additives, recovers the LD bacteria in about 60% of cases. Growth occurs after 2 to 7 days. Blood, sputum, drainage from dialysis fistulas, and other body fluids (such as pleural fluid) should all be cultured if possible.[29] The cost of culture is about $20. Occasionally, cultures will be positive even when direct fluorescent antibody tests were negative. Patients with nonproductive coughs may present problems. Only 0.5 ml of sputum is needed for culture and immunology. If this fails, transtracheal aspirate or bronchoscopic lavage may be obtained for diagnostic cultures; however, saline solutions should be avoided since they may inhibit legionella.[30]

**Indirect fluorescent antibody** (IFA) diagnosis of LD is based on four-fold increase in serum antibody to a titer of 1:128 or greater. This method is well standardized for disease caused by *L pneumophila* serogroup 1, demonstrating a specificity of 95% and sensitivity of about 70%, but is not yet standardized for other legionella species.[30] Cost of IFA is about $35. Antibody titer elevations occur over a period of four weeks so that convalescent titers are necessary in order to document a diagnostic rise. A presumptive diagnosis of legionella infection can be made with a titer over 1:256 when associated with clinical illness.[13] It should be remembered, however, that previous legionella exposure may cause persistent titer elevations years after exposure, complicating efforts to diagnose acute infection.

Other serum antibody tests, such as enzyme-linked immunosorbent assays (ELISA) promise new alternatives in diagnosis and may be widely available in the near future.

## Treatment of Legionnaires' Disease

Treatment begins with general supportive measures and discontinuation of steroids and immunosuppressants if possible.[13,14]

The antibiotic of choice for Legionnaires' disease is erythromycin, 3 to 4 gm/day intravenously for the first 7 days, followed by 2 gm/day orally for 14 days.[13,14] Few relapses have been reported when this regimen was followed. Rifampin has been shown to be a very effective adjunct to erythromycin, but should be avoided as a single agent as resistance may emerge.[13,14] The advantage in using either of these two drugs is that they are concentrated within alveolar macrophages where many LD bacteria are typically located.

Penicillins, cephalosporins, and aminoglycosides have no role in the treatment of Legionnaires' disease.[20] Progression of disease despite therapeutic doses of these medicines has been demonstrated repeatedly.

If erythromycin cannot be given, an alternative is doxycycline, preferred over tetracycline for its better lipid solubility. An initial dose of 200 mg, then 100 mg 12 hours later, then 100 mg a day, is effective. Rifampin may be used simultaneously for severely ill patients.[13]

Clinical response to erythromycin therapy is demonstrated by subjective improvement within 24 to 48 hours after therapy is initiated. Fever subsides quickly, although radiographic pulmonary consolidations may persist for weeks.

## Prognosis

The prognosis and case-fatality rate is dependent upon the presence of underlying disease and appropriate use of antibiotics. Case fatality rates range from 7% in nonimmunosuppressed patients treated with erythromycin to 80% in immunosuppressed patients who did not receive erythromycin. The overall case fatality rate is about 20% in patients with underlying disease managed with appropriate measures.[13,14]

## Faculty Comment

(Douglas P. Fine, MD, Professor of Medicine and Chief of the Infectious Disease Section, University of Oklahoma Department of Medicine, comments on the relevance of *L pneumophila*.)

Legionnaires' disease is an important cause of both nosocomial and community-acquired pneumonia. It should be considered especially in a middle-aged or elderly smoker who develops an atypical pneumonia, ie, somewhat insidious onset, prominent cough with relatively scant sputum, only moderate numbers of organisms on sputum gram stain, and patchy pneumonic infiltrates. Neurological and gastrointestinal symptoms may be important clues. The most clinically useful diagnostic test is direct fluorescent antibody staining of sputum; culture is useful if available; serology is confirmatory in retrospect. Erythromycin is the antimicrobial of choice.

*Acknowledgment* Special thanks to Drs P. A. McKee, D. P. Fine, and L. Slater for making this paper a reality, and to Ms B. Clarke for her conscientious preparation.

Answers to Test Your Knowledge:
1. c; 2. e; 3. b; 4. a; 5. d

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## Coming in April . . .

Manuscripts being considered for publication in April include an in vitro comparison of cefotetan with other antibiotics, a report on augmentation enterocystoplasty in children with myelomeningocele, and a commentary on barber surgeon farmers.

# Current Concepts in Nutrition: Enteral Tube Feeding

STEPHEN R. NEWMARK, MD; CHRISTINE KOELZER, RN; MARGARET HALEY MCCOWN, RD

**The proper management of patients supported with enteral tube feeding can be facilitated with the use of a tube feeding protocol. This protocol will provide for regular nursing orders as well as the proper laboratory determinations needed to maintain a normal metabolic profile.**

It is estimated that 10% to 20% of all hospitalized patients have protein-calorie malnutrition as a primary or secondary diagnosis.[1-5] Nutritional deficiency states may increase the risk of severe infections, decrease cardiac function, and retard wound healing.[6-9] In addition, there are multiple adverse effects on renal gastroenterological, hepatic, pulmonary, dermatological, neurological, and musculoskeletal organ systems.[6-10] In appropriately selected patients, nutritional support may reverse or prevent many of the complications associated with nutritional deficiency. It is the purpose of this review to discuss the current modalities of enteral tube feeding for the hospitalized patient.

## Selection of Candidates for Tube Feeding

If a patient is unable to ingest sufficient calories to prevent clinically significant malnutrition, enteral tube feeding can be selected as a nutritional support system provided the gastrointestinal tract is functional. Frequent disease categories associated with protein-calorie malnutrition that may be included are: (1) acute and chronic infections, (2) multiple trauma, (3) respiratory insufficiency, (4) neurosurgical injuries, (5) patients receiving radiation or chemotherapy for advanced cancer, (6) neurological disorders that prevent voluntary ingestion of food, (7) eating disorders associated with psychiatric illness, and (8) upper gastrointestinal obstruction in which a feeding enterostomy has been surgically placed distal to the obstruction.

## Enteral Feeding System

Currently available enteral feeding tubes are composed of silicone elastomer or polyurethane structural components and are reasonably flexible, with an acceptable size (8 French) adding to patient comfort. Most feeding tubes have a metal stylus to aid in intubation: after successful placement of the tube, the stylus can be removed. The tips of most feeding tubes are radio-opaque and can be demonstrated on an x-ray to determine the placement. It is recommended that the placement of all tubes be ascertained by gastric suction, by ausculation over the gastric fundus, or by x-ray. If the tube is designed to pass through the pylorus into the duodenum, an x-ray will be necessary to confirm the position in the small bowel.[10]

Although tube feedings can be delivered by gravity flow, it is generally desirable to control the rate of flow by means of a feeding pump. The use of a feeding pump can also regulate the flow of the formula in patients who require strict volume control, as well as provide an alarm system should unexpected resistance to flow be encountered.

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Usually the tube feeding is delivered as a continual infusion over 24 hours; this facilitates delivery and lessens the chance of gastrointestinal pooling which is encountered more often in "bolus" delivery. As a general rule, aspiration through the feeding tube with a syringe is performed once each shift (every eight hours) to detect any possible pooling of formula within the gastrointestinal tract which may lead to possible distention or aspiration. Generally, the residual volume detected by aspiration should be less than 20 to 30 ml. If more than 100 ml are aspirated, the flow rate of the tube feeding is decreased or the tube feeding is discontinued until the patency of the gastrointestinal tract is assessed, or associated motility disorders are corrected.

## Tube Feeding Formulas

There are multiple enteral tube feeding products available for the physician. Because of economic concerns, few hospitals will be able to stock a majority of existing formulas; therefore, it is wise for the physician to become familiar with the solutions that are available in his or her own practice environment. Examples of different types of tube feeding formulas are listed in the table.

The isotonic formulas (Isocal, Ensure, Osmolite) can be used as standard tube feeding solutions. These formulas can be initiated at full strength beginning at 50 ml per hour and increasing by 25 ml per hour each day until the desired infusion rate is obtained. An infusion rate of 125 ml per hour will provide approximately 3000 calories, 100 grams of protein, 50 to 60 mEq of sodium, and 70 to 90 mEq of potassium per day. The daily recommended adult allowance for vitamins and other minerals will be satisfied by an infusion rate of 75 to 100 ml per hour (1800 to 2400 ml per day).

Isocal HCN can be used in clinical settings in which volume must be controlled. In this instance, Isocal HCN will supply 2000 calories, 75 grams of protein, 35 mEq of sodium, and 36 mEq of potassium per liter. Conversely, Isocal HCN can be utilized when increased amounts of calories and protein must be supplied to patients with exceptionally high catabolism such as certain patients with internal injuries or multisystemic trauma with infection. Sodium and potassium can be added directly to the solution if necessary. Because Isocal HCN is hypertonic, it is best initiated at half-strength and slowly increased in tonicity as the desired flow rate is obtained.

Certain formulas containing peptides as protein sources (Vital HN) or crystalline amino acids (Vivonex HN and Vivonex TEN) can be used when minimum residue is desired or when there may be impaired digestion or absorption of intact proteins secondary to inflammatory bowel disease or the short bowel syndrome. It should be emphasized that some investigators have demonstrated that solutions with intact proteins can also be metabolized in the above disorders and that other nutritional components such as fat and certain carbohydrates may be more important determinants of the efficacy of the enteral formula. An additional advantage of the peptide and crystalline amino acid solutions is decreased viscosity which allows their use in narrow-caliber access routes such as a needle catheter jejunostomy.[3 & 4] The use of an enteral tube feeding protocol will standardize nursing orders and facilitate delivery of the feeding solution.

Table. Tube Feeding Formulas

| Formula | Osmolality (Mosm/L) | Protein (G/L) | Calories (Kcal/L) | Sodium (mEq/l) | Potassium (mEq/L) |
|---|---|---|---|---|---|
| Isocal | 300 | 34 | 1000 | 23 | 34 |
| Ensure | 300 | 37 | 1000 | 32 | 33 |
| Osmolite HN | 300 | 46 | 1000 | 40 | 40 |
| Isocal HCN | 590 | 75 | 2000 | 35 | 36 |
| Vital HN | 450 | 42 | 1000 | 17 | 30 |
| Vivonex TEN | 532 | 38 | 1000 | 20 | 20 |
| Vivonex HN | 810 | 46 | 1000 | 23 | 30 |

## Complications of Enteral Tube Feeding

Problems encountered with enteral tube feeding usually consist of mechanical difficulties with the feeding tube or pump, gastrointestinal dysfunction, or metabolic abnormalities secondary to the formula infusion.[10]

**1. Mechanical difficulties** If the feeding tube becomes occluded with an inspissated solution, aspiration with a syringe or flushing with cranberry juice may clear the tube. If the tube remains occluded, it will need to be withdrawn and a new feeding catheter reinserted. If the catheter becomes dislodged or migrates to an esophageal location, the feeding tube should be withdrawn and a new catheter inserted.

**2. Gastrointestinal dysfunction** Diarrhea is the most frequent abnormality that is encountered with tube feeding. This usually can be controlled by regulating the flow rate or decreasing the osmolality of the solution. Occasionally, bowel overgrowth with candida is responsible and can be treated with Mycostatin instilled via the tube. Bacterial-induced

enterocolitis also may occasionally be encountered; this is usually secondary to *Clostrida difficile* and can be treated with bowel rest and appropriate antibiotics. It is also important to recognize that intermittent bowel obstruction may be a cause of diarrhea and abdominal distention.

**3. Metabolic abnormalities** It is important to include periodic biochemical testing for patients supported with enteral tube feeding to assess and monitor possible metabolic changes. Hyponatremia was encountered in 31% of the patients in one series and was secondary to overhydration in all cases. Hypokalemia and hypophosphatemia were encountered in 10% and 30% of the cases respectively and were secondary to diuretic and/or insulin use.[11] Hyperglycemia was noted in 40% of the cases and was associated with infection or multisystemic illness. Insulin administration usually as NPH or Lente given every 12 to 24 hours was necessary to maintain the glucose levels <200mg%. It is important to note that insulin doses need to be readjusted when the feeding rate is decreased or the enteral feeding system is discontinued.

Enteral tube feeding can be an extremely effective method to provide nutrients and fluids to patients who cannot ingest food and water to maintain metabolic homeostasis. The utilization of a standard protocol will facilitate the delivery of the formula to the hospitalized patient.

Although costs and charges for enteral tube feeding will vary depending upon individual products and hospital policy, the cost of 1000 calories (1000 ml) by enteral infusion of a standard solution usually ranges between $1 and $5 compared to 1000 ml of dextrose/water by intravenous infusion, which ranges between $10 and $20 and to total parenteral nutrition (TPN), which ranges between $60 and $90 per 1000 calories. Specialized tube feeding solutions, such as solutions containing crystalline amino acids or solutions intended for hepatic or renal support, are more costly and may approach the cost of parenteral solutions.

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News from
the Oklahoma State
Department of Health

# Lead Poisoning Prevention

In 1985 the Centers for Disease Control (CDC) issued recommendations for blood lead levels and lead toxicity to be used in classifying children for follow-up and treatment of lead poisoning.

CDC's guidelines for an elevated blood level reflects excessive absorption of lead at a confirmed concentration of 25 micrograms of lead per deciliter of whole blood (ug/dl). This new level is based on epidemiologic, clinical, and experimental evidence which indicates that lead is toxic at levels previously believed to be acceptable. The previous safe level was believed to be 30 ug/dl of whole blood.

CDC's recommendation for a lead toxicity level is an elevated blood lead level with an erythrocyte protoporphyrin (EP) level in whole blood of 35 ug/dl, or greater.

A survey conducted by the Oklahoma State Department of Health in 1985 screened 1,073 children and identified 15 children with elevated lead levels. The Oklahoma State Department of Health acknowledges the CDC lead level recommendations and suggests that components of a successful lead-poisoning prevention program include screening, diagnosis, treatment, and follow-up, as well as environmental intervention for children with elevated blood lead levels. The ultimate goal is to remove lead from the environment of children before it enters their bodies.

| DISEASE | December 1986 | TOTAL TO DATE | | |
|---|---|---|---|---|
| | | This Year | Last Year | 5 Yr. Avg. |
| AMEBIASIS | 2 | 9 | 19 | 15 |
| CAMPYLOBACTER INFECTIONS | 12 | 258 | 297 | — |
| ENCEPHALITIS, INFECTIOUS | 1 | 22 | 29 | 32 |
| GIARDIA INFECTIONS | 20 | 245 | 326 | — |
| GONORRHEA (Use ODH Form 228) | 972 | 12572 | 13005 | 14651 |
| HAEMOPHILUS INFLUENZAE INVASIVE DISEASE | 22 | 249 | 276 | — |
| HEPATITIS A | 20 | 341 | 471 | 598 |
| HEPATITIS B | 23 | 217 | 244 | 285 |
| HEPATITIS, NON-A NON-B | 2 | 61 | 75 | — |
| HEPATITIS UNSPECIFIED | 3 | 49 | 84 | 182 |
| MEASLES (RUBEOLA) | 0 | 39 | 1 | 9 |
| MENINGITIS, ASEPTIC | 22 | 150 | 146 | 194 |
| MENINGITIS, BACTERIAL (non-meningococcal, non H. Influenzae) | 22 | 86 | 60 | 67 |
| MENINGOCOCCAL INFECTIONS | 3 | 32 | 34 | 36 |
| PERTUSSIS | 8 | 134 | 197 | 163 |
| RABIES (Animal) | 4 | 62 | 111 | 146 |
| ROCKY MOUNTAIN SPOTTED FEVER | 1 | 104 | 93 | 123 |
| RUBELLA | 0 | 0 | 1 | 1 |
| SALMONELLA INFECTIONS | 30 | 480 | 465 | 483 |
| SHIGELLA INFECTIONS | 22 | 227 | 283 | 344 |
| SYPHILIS (Use ODH Form 228) | 15 | 166 | 215 | 204 |
| TETANUS | 0 | 1 | 1 | 1 |
| TUBERCULOSIS | 17 | 254 | 248 | 294 |
| TULAREMIA | 1 | 13 | 19 | 31 |
| TYPHOID FEVER | 0 | 2 | 2 | 3 |

| Diseases of Low Frequency | Total to Date This Year |
|---|---|
| ACQUIRED IMMUNE DEFICIENCY SYNDROME | 48 |
| BRUCELLOSIS | 4 |
| LEGIONNAIRES DISEASE | 20 |
| MALARIA | 12 |
| REYE SYNDROME | 5 |
| TOXIC SHOCK SYNDROME | 37 |

*Disease often unrecognized*

# State scleroderma group ready to help physicians and patients

In July 1986 the Oklahoma chapter of the United Scleroderma Foundation was established. It is intended to be a support group for persons with this disease or related conditions, as well as a respository of resources and information. It can augment a patient's own efforts at self-help and encourage a specific referral where indicated. In order for these efforts to be effective, however, the disease must be recognized by patients and physicians.

Physicians are encouraged to be aware of this disease and, if they have patients with scleroderma, to let them know about the Oklahoma chapter of the United Scleroderma Foundation. Information can be obtained from (405) 373-2914 or (405) 360-6160.

Systemic sclerosis is a generalized disorder of connective tissue characterized by proliferative vascular lesions, obliterative microvascular lesions resulting in fibrosis, degenerative changes, and atrophy of many organs. Scleroderma (literally meaning "hard skin") was the original name and is the hallmark of the disease. It is present in 95% of cases. This is the second phase of cutaneous involvement, in which the skin takes on the classic hidebound appearance due to dense collagen infiltration in the dermis and atrophy of the epidermis and loss of skin appendages. The first phase of skin involvement is characterized by edema and puffy fingers. The third phase involves a return to more normal-appearing skin. However, it is important to recognize that many other organs are involved in systemic sclerosis. There are even a few cases of gastrointestinal involvement without skin disease (systemic sclerosis sine scleroderma). Occasionally, localized forms of the disease, such as morphea or linear scleroderma without systemic involvement, occur.

Systemic sclerosis is really a multisystem disease. The earlier term, progressive systemic sclerosis, is not used now because the rate of progression of the disease in these various organ systems is quite variable. The disease may be stable in any given organ system over a period of many years. In addition to cutaneous involvement, it is quite common to have vascular changes with Raynaud's phenomenon, which occurs in approximately 90% of the cases. Raynaud's phenomenon occurs alone and in other conditions, but abnormalities of microvascular structures may predict the development of systemic sclerosis in patients with Raynaud's phenomenon. Capillaries can be directly observed with low-power magnification in the nail folds. Abnormalities including dilated capillary

*(continued on p 168)*

**OSMA**

**Physician Recovery Hotline**

**(405) 691-7318**

# CALL FOR RESOLUTIONS

All resolutions to be presented to the Oklahoma State Medical Association House of Delegates Annual Meeting must be received in the OSMA executive offices no later than thirty (30) days prior to the meeting. This year's meeting will be held May 1-3, 1987, at the Sheraton-Century Center in Oklahoma City.

County medical societies or individuals wishing to submit resolutions should mail them to the OSMA, 601 Northwest Expressway, Oklahoma City, OK 73118. Should you need assistance in drafting such resolutions, please contact the executive offices at (405) 843-9571 or 1-800-522-9452.

SUBMIT YOUR RESOLUTIONS
ON OR BEFORE
APRIL 1, 1987

1987 OSMA
ANNUAL MEETING

## Scleroderma group (continued)

loops and capillary drop-out have been observed in patients with systemic sclerosis. There is a strong correlation between these capillary nail fold findings and visceral involvement with systemic sclerosis. Another predictor of systemic sclerosis in patients with Raynaud's phenomenon is the presence of anticentromere antibodies seen on ANA preparations on HEp-2 cells.

Gastrointestinal involvement is very common, and esophageal hypomotility is seen in 90% of patients. Pulmonary involvement includes pleural manifestations as well as the development of pulmonary hypertension and diffuse interstitial lung disease. Cardiac involvement includes pericarditis, pericardial effusion and rarely, myocardial fibrosis. Complete heart block can be seen. Skeletal muscle involvement is not uncommon. A clinical picture similar to acute polymyositis is seen. It is one of the few indications for corticosteroids in the disease. A seronegative symmetrical polyarthritis similar to rheumatoid disease can be seen. This is usually not very prominent and is nonerosive and nondeforming. Renal involvement is actually fairly common, with histologic changes apparent in 80% of autopsy patients. However, only one-third to one-half of the patients develop clinical manifestations. Fewer than 10% develop the dreaded scleroderma kidney, with rapidly progressive renal failure. It is important to recognize this development, though, because newer forms of treatment, particularly those with angiotensin converting enzyme inhibitors and dialysis, can be life-saving.

The CREST syndrome has been recognized in the past as a variant of this disease. It consists of *c*alcinosis, *R*aynaud's phenomenon, *e*sophageal hypomotility, *s*clerodactyly, and *t*elangiectasias. It also can include pulmonary fibrosis, pulmonary hypertension, biliary cirrhosis, and trigeminal neuralgia. It is currently thought that this is not just a mild form of the disease but a subset of patients in which visceral involvement is delayed to the second or third decade.

Currently there is no one form of treatment that is effective for all manifestations of the disease. Many drugs and regimens have been tried. Only D-penicillamine has offered some hope of slowing the progression of cutaneous involvement. However, this still remains controversial. Treatment, in general, is directed towards specific target system involvement.

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*Biggest hazard: accidental needle-sticks*

# CDC data confirms AIDS risk to health care workers is low

A review of federal AIDS data confirms that health care workers face little occupational risk of human immunodeficiency virus (HIV) infection, says a report in the *Journal of the American Medical Association*.

Alan R. Lifson, MD, MPH, of the Centers for Disease Control (CDC), Atlanta, and colleagues studied data reported to the CDC on AIDS patients as of May 1, 1986. For health care workers with AIDS, the researchers say, there were no specific occupational exposures that could be implicated as an HIV infection source.

The CDC had occupational data on about 17,000 of 20,000 adult AIDS patients reported as of May 1. Of these, 922 were listed as health care workers, all but 88 of whom were classified as being in groups at high risk for AIDS.

Ten of the 88 were from countries where many AIDS cases have been linked to heterosexual contact and receipt of blood transfusions; a risk history or other data were incomplete or unavailable on 17 more. Follow-up data on the remaining 61 workers first categorized as having no identifiable risk resulted in 44 being reclassified — meaning 95% of the health care workers with AIDS studied were listed in high-risk groups overall, the study reports.

For workers with no identifiable risk, the researchers sought to document specific occupational incidents that might have caused the infection. "Although the possibility of infection through anonymous or undocumented exposures cannot be eliminated," they say, "no specific, conclusive link was found."

The study says risk factors may not be identified in AIDS patients for various reasons, such as incomplete information, including that which patients might be reluctant to disclose, and the fact that some patients might not have AIDS even though their cases meet definition criteria. Unrecognized heterosexual transmission could account for some of these cases as well.

Although the CDC could pinpoint no occupational exposures for the health care workers studied, several reports from the US and England suggest HIV can be transmitted this way. Several studies assessing this risk are under way, one a CDC study of nearly 1,000 health care workers exposed to blood or other body fluids from AIDS patients. Of 451 people in this study tested, only two were HIV antibody-positive says the CDC report, noting "other studies have also concluded that the risk transmission of HIV from infected persons to health care workers is low. . . . Our surveillance data support (this) conclusion."

The CDC researchers conclude that adherence to recommended infection control guidelines should further decrease any occupational risk. This is emphasized in an accompanying editorial by Michael D. Decker, MD, MPH, and William Schaffner, MD, of the Vanderbilt University School of Medicine, Nashville.

Some of the reports of occupational HIV infection among health workers have involved accidental punctures with contaminated needles. The editorial says this is "the most important occupational source of risk of HIV infection among health care workers," even though it rarely causes HIV transmission.

Transmission through skin contact with highly contaminated patient body fluids also has been reported in isolated cases, but this risk "either is so small or so easily averted by routine infection-control precautions that thus far there is no evidence of HIV having been transmitted (this way) to a professional health care worker," the editorial says.

It concludes, "The risk of occupational transmission of AIDS to health care workers can be essentially eliminated by scrupulous attention to established infection-control and needle-stick recommendations."

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# Therapeutic substitution of drugs usurps MDs' prerogative

Therapeutic substitution, in which pharmacists may substitute for a drug prescribed by a physician any other drug they believe has similar therapeutic effect, is a "usurpation of (physicians') professional prerogatives," says an editorial in the *Journal of the American Medical Association.*

Therapeutic substitution of drugs by pharmacists is vigorously opposed by the AMA, according to John C. Ballin, PhD, Director Emeritus of the AMA's Division of Drugs and Technology.

Therapeutic substitution is a "giant step beyond generic substitution because the drug dispensed is not another version of the same drug, but *an entirely different chemical entity,"* says Ballin. In therapeutic substitution, a "pharmacist can substitute any drug believed by the pharmacist to have a similar therapeutic effect as the drug prescribed by the physician," he says.

Ballin mentions four states (Washington, California, Iowa, and Florida) that have made inroads into therapeutic substitution or independent prescribing, pointing out that physicians may not be aware of the extent of the situation. Physicians do, however, share a consensus that opposes the practice. Their opposition, he feels, is "nonnegotiable."

Calling prescribing of drugs "an integral part of the practice of medicine," the author cites the importance of patient history, physical examination, diagnosis of various disease states, and overall development of a treatment plan as crucial to deciding on the optimum drug choice for a patient.

Thorough evaluation of all clinical and psychosocial aspects of a patient's medical needs requires the medical training of a physician. The elderly are especially susceptible to miscues in prescribing medication. They frequently suffer from multiple diseases, may be taking more than one medication, and often have frail biological systems.

A combination of forces has conspired to motivate state pharmaceutical associations to lobby for legislation allowing for therapeutic substitution, Ballin says. He sees economic pressures as being at the base of these forces. Many PPOs and HMOs that dispense drugs to their patients or have contracts with large chains may be forcing community pharmacists to lower operating costs, he notes. Increasing measures such as these toward cost containment in health care create pressures on community pharmacists.

Ballin says the physician's point of view is best summarized in the first chapter of the drug text *AMA Drug Evaluation:* "The prescription of a drug represents the culmination of a deliberative process between physician and patient aimed at the prevention, amelioration or elimination of a disease or disorder." Ballin concludes: "Only a medical education is adequate to prepare one to evaluate all the scientific and psychosocial data needed to make a sound therapeutic decision."


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*Now about one in five live births*

# Defensive medicine? Cesarean section rate still increasing

The rate of cesarean section deliveries in the US shows no sign of leveling off or declining, despite a 1980 government conference scrutinizing the rapid increase in the frequency of the surgery, says a study in the *Journal of the American Medical Association*.

There has been a sharp jump in the practice of giving women trials of labor after a previous C-section, says the study by Patricia H. Shiono, PhD, of the National Institute of Child Health and Human Development (NICHD), Bethesda, and colleagues. "However, these changes have not been sufficient to stem the rising cesarean delivery rates (now about one-in-five live births)," they say. "This (study) and other national surveys provide no evidence that the cesarean delivery rates are leveling off or decreasing."

The study was designed to assess the impact of a 1980 NICHD Consensus Development Conference on Cesarean Childbirth, called to focus attention on the increasing use of C-sections in the US. Conference recommendations included increased use of trials of labor for selected women who had previous cesareans, careful scrutiny of the criteria for the diagnosis of abnormal labor, and vaginal delivery of uncomplicated term breech infants.

NICHD researchers mailed questionnaires to 538 hospitals, asking about their cesarean and trial-of-labor practices. Nearly 90% responded.

The survey revealed the C-section rate increased from 14.1% of births in 1979 to 19% in 1984. This was about 2% lower than earlier best estimates of the frequency of the surgery, the NICHD study says, a difference probably due to the differences in methodology.

Trials of labor increased almost four-fold, from 2.1% in 1979 to 8% in 1980, with the range of rates closely correlated with the size of the hospital's delivery service, the study reports. However, the percentage of hospitals not allowing this practice

*(continued on p 175)*


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*Head lice don't discriminate*

# School can go to child's head — and to the rest of the family

What do the first week of school, the week following Christmas vacation, and the last week of school have in common? Pediculosis or head lice. More outbreaks of head lice occur during these times than at any other.

The head louse, or *Pediculus humanus capitis,* is a very small insect, about the size of a sesame seed, that lives in human hair. Head lice (more than one louse) live and breed only on a human host. Without a human host, lice usually live 12 to 15 hours. They cannot live on cats, dogs, or other household pets.

The primary symptom of head lice infestation is the itching that occurs when lice bite and suck blood from the scalp. Children seen scratching their heads frequently should be checked for lice immediately. As the infested individual scratches, secondary bacterial infections may occur at the scratch site, particularly in the case of children, whose hands may not be clean.

Head lice are difficult to see because they move quickly and shy away from light. Diagnosis is sometimes made on the basis of finding nits or eggs. Nits may be found anywhere in the hair, but are frequently found at the nape of the neck, behind the ears and on the crown. Often a magnifying lens is helpful in identifying nits.

Lice cannot fly or jump from one place to another. They are transmitted by direct contact with an infected individual or by sharing items of clothing or grooming aids with an infected individual. It is *not true* that only unclean people get head lice; *anyone can get head lice!*

Children should be warned not to share such items as combs, brushes, barrettes, hair ribbons, headbands, etc with others. Children participating

(continued on p 176)

## Cesarean section (continued)

remains high — more than half, it notes. More than 50% of trials of labor were successful, the researchers add.

More primary cesareans were due to fetal distress in 1984 (21%) than 1979 (14%), but the researchers say it was not clear to what extent the diagnosis of fetal distress may have changed because of fetal monitoring or other reasons. The percentage of cesareans performed because of abnormal labor or breech presentation remained virtually unchanged over the period.

The researchers can't explain exactly why the cesarean rate continues to rise. Although many point to medical-legal issues, the study notes, the 1980 NICHD consensus conference concluded that " 'although commonly cited, there is no evidence that fear of litigation and the possible consequent practice of defensive medicine is a major cause of the increased cesarean birth rate.'

"Though formal evidence is still lacking, the perception that concern about litigation tilts borderline cases toward abdominal delivery appears to have become increasingly widespread over the last six years," the researchers conclude.

## In Memoriam

### 1986

| | |
|---|---|
| *Alexander Poston, MD* | *January 3* |
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| *Edward L. Leonard, MD* | *February 14* |
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| *Fred D. Switzer, MD* | *May 10* |
| *John D. Jennings, MD* | *May 12* |
| *Phillip Wade Jones, MD* | *May 18* |
| *Herbert L. Owen, MD* | *May 28* |
| *Marianne Elsbeth Kosbab, MD* | *June 13* |
| *William W. Rucks, Jr., MD* | *June 27* |
| *Ralph A. Smith, MD* | *July 27* |
| *Howard D. Tuttle, MD* | *August 3* |
| *Welborn W. Sanger, MD* | *September 19* |
| *William Carl Ewell, MD* | *September 20* |
| *Marcella Steel, MD* | *October 1* |
| *Terry Dwight Leming, MD* | *October 13* |
| *William Pat Fite, Jr., MD* | *October 30* |
| *Samuel Jackson McDaniel, MD* | *November 2* |
| *Iron Hawthorne Nelson, MD* | *November 12* |
| *John Robert Walter Spencer, MD* | *December 4* |

## Head lice (continued)

in team sports should be checked frequently for lice since batting helmets, football helmets and other kinds of headgear are often passed from player to player. Children should also be advised not to stack or pile up sweaters, coats, etc, and to hang coats so they do not touch. Wearing clothing recently worn by an infected person (especially head garments) or lying on infested furniture, carpeting, or bedding can also produce a case of lice. For that reason, *all* family members should be examined when one has lice, and everyone showing signs of infestation should be treated at the same time.

A single individual will usually harbor fewer than 20 lice, although infestations with hundreds have been reported. The length of one's hair has not been proven to be a factor. Girls do seem to have a higher incidence of lice than boys, but it is thought that this is due to their more intimate play patterns and to their closer contact with younger children. Black children have a far lower incidence of head lice than either white or Oriental children.

Treatment for lice is a two-step process. First, a special head lice product must be used to kill the crawling lice. Frequent bathing and shampooing will not prevent head lice nor eliminate them once they are established. Using a blow dryer on the hair will also have little or no effect on lice.

A pharmacist or the health department will help you obtain the appropriate product. Whatever pediculicidal product is used, package directions should be followed explicitly and all safety precautions observed.

Lice and nits on children under the age of two years should be removed manually without the use of a commercial lice product.

Head lice products should always be applied by an adult. They should not be used in the bathtub or shower or immediately following a hot bath. Parents should treat children by leaning them over the sink to confine the product to the head and neck areas. Be sure to protect the child's eyes by holding a dry washcloth or towel over them.

The second step of the treatment involves the

(continued on p 177)


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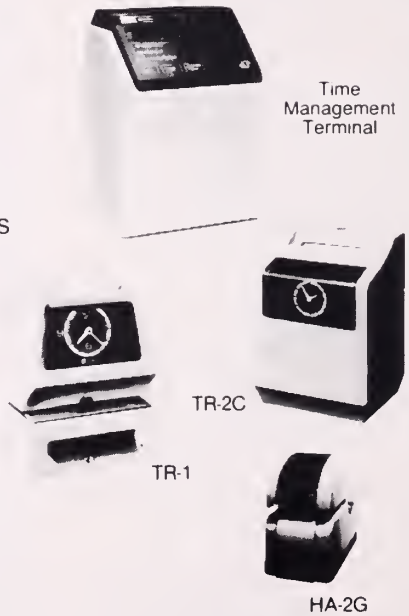


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**Clinical Hematology.** 8th Edition. By Maxwell M. Wintrobe, G. Richard Lee, Dane R. Boggs, Thomas C. Bithell, John Foerster, John W. Athens, and John N. Lukens. Philadelphia: Lea & Febiger, 1981. Pp 2021, illustrated, price $102.00.

The first edition of this classic was published in 1942. At that time hematology was being transformed from the morphologic discipline it had become to one that was beginning to rely increasingly on the methodology of physiology, biochemistry, and experimental medicine. As pointed out in the preface, hematology encompasses many aspects of cytology, biochemistry, biophysics, molecular and cellular biology, genetics, immunology, pharmacology, and oncology, all of which must be discussed in a textbook on this subject. This book, then, is a comprehensive compilation of current knowledge in the fields which traditionally make up hematology.

There have been certain important changes since the seventh edition was published some nine years ago. One of the most important is the addition of Dr John Lukens, an authority in pediatric hematology-oncology, to the list of authors. A section limited to neoplastic hematologic disorders has been added. Other chapters have been rearranged. The book has been expanded by 126 pages, and there has been an updating of certain portions of the appendix.

The format of the book remains essentially the same as before — each chapter is followed by an index organized by subject headings. At the conclusion of the text there is a comprehensive bibliography. As before, the book is generously filled with illustrations, graphs, and other drawings and photomicrographs. In addition there are 20 color plates illustrating normal and abnormal cells of the blood and hematopoietic system. This edition is only 25 pages longer than the last; to accommodate the additions the text is printed in smaller type.

The eighth edition contains further improvements in this classic text.

*Harris D. Riley, Jr., MD*
*Oklahoma City*

## Head lice (continued)

removal of all the eggs or nits. Because nits are firmly attached to the hair shaft, a special combing tool is needed to help remove them. Nits that cannot be combed out should be removed by hand, using the fingernails, tweezers, or scissors.

Parents are often instructed to fumigate bedding, carpeting, and furniture with commercially available lice sprays. However, both the Centers for Disease Control and the National Pediculosis Association *strongly discourage the use of sprays,* as they are of highrisk and questionable benefit. As an alternative to spraying, careful vacuuming of the home, including mattresses and upholstered furniture, and school, eg, tumbling mats, can be done. Bed linens and recently worn clothing can be washed and dried in the dryer, vacuumed, or bagged in plastic for two weeks.

With public attention currently focused on the quality of children's health care, no camp, day care, or school health program is complete without addressing pediculosis. The negative effects suffered by the child with this highly communicable condition range from physical discomfort and social stigma to loss of valuable class time to repeated exposures to pesticides. Ultimately, families and whole communities are victimized by the anxiety, frustration, and anger that often accompany lice outbreaks.

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# LOSS PREVENTION SEMINAR ATTENDANCE MANDATORY

Attendance at an OSMA-PLICO sponsored Loss Prevention Seminar is now mandatory for all PLICO-insured physicians at least once every three years. **If a physician has never attended a seminar, he or she must attend one during 1987.** If a physician has not attended a program since 1984, he or she must attend this year, also.

The change making seminar attendance mandatory was implemented by the PLICO Board of Directors in late 1985, and a special endorsement outlining the requirement was included in all 1986 PLICO professional liability insurance policies. Any physician needing to attend in 1987, and failing to do so, will not be eligible for renewal of his or her PLICO professional liability insurance for calendar year 1988.

A Seminar Registration Form is located at the bottom of this page and a schedule of upcoming seminars is included. The registration form may be used for any seminar, but please **specify the date** you would like to attend. It is advisable to pre-register for the seminar you would like to attend.

## OSMA-PLICO 1987 LOSS PREVENTION SEMINARS

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| August 8 | Sat. 2-5 p.m. | WOODWARD Park Inn |
| September 19 | Sat. 8:30 a.m.-11:30 a.m. | McALESTER Holiday Inn |
| September 26 | Sat. 8:30 a.m.-11:30 a.m. | OKLAHOMA CITY Lincoln Plaza |

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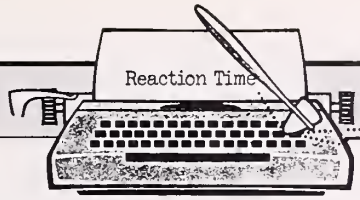


## "Trash such articles," says Clinton physician

*To the editor:* I have always enjoyed your articles in the state medical journal, and usually always read the JOURNAL from cover to cover. I could not help but write this letter regarding the commentary by Joe D. Haines, Jr., MD, [JOSMA, Dec 86] regarding pharmaceutical representatives.

It is my feeling that it is mostly garbage and completely out of character. There are very many top-notch representatives, and many of them are my friends. I resent the statement that they should all be done away with.

In these days and times, it amazes me that someone would promote unemployment when the unemployed are already suffering so much. There are still some of us physicians who have been in medicine for forty-such years who appreciate a break in seeing patients to discuss hunting and fishing, and other forms of relaxation with the drug representatives.

As for the "freebies" that he discussed, I feel they come in very useful for people who cannot afford the cost of medicine today. My suggestion is that you trash such articles as this in the future.

*J. Harold Tisdal, MD*
*Clinton*

## Dewey doctor defends pharmaceutical representatives

*To the editor:* To say that I was taken aback on reading Dr Haines' article [JOSMA, Dec 86] condemning all drug reps is to put it mildly.

Perhaps a surly attitude begets a surly response. Anyway, I can only say that the pharmaceutical representatives that I encounter are, almost to a man (or woman) considerate, polite and very understanding when I am delayed for an appointment. And yes, I do set aside time in my office to see these gentlemen (ladies). It's a time I thoroughly enjoy, and I learn a lot, not only about medicine but about that outside world that is continually encroaching more and more on the practice of medicine.

I cannot fault Dr Haines for wishing to do nothing but read medical journals. It is a manner much to be desired. But unfortunately it takes a lot of time. Time, we are told, is what we must save with computers, dictation, better scheduling, etc. All of this doesn't really add up to "lazy."

Incidentally, many of our state and local meetings are at least partially dependent on the largesse of the pharmaceutical companies. Dr Haines may consider all the pharmaceutical booths at our conventions the work of the devil, but most of us enjoy the displays as well as the evening events provided to us.

Last and perhaps most important is the appreciation shown by people who receive some of their prescription medicine free. These things are very expensive at the pharmacy and a few extra for nothing means a lot.

Oh well, I'm sure I haven't changed Dr Haines' mind, but I really do like these people and I hope they consider me their friend. I do not use every one of the products they present but I probably will use the products of those who call on me in preference to those who don't because it means a lot monetarily to my patients. The "knick-knacks" too, are intriguing and some quite practical.

So as I casually prescribe all these medical miracles I do thank the pharmaceutical industry and their representatives who make it possible. And, oh yeah, stop by my place anytime.

*John R. Smithson, MD*
*Dewey*

## Confer's experience "exactly the opposite of Dr Haines"

*To the editor:* After reading Joe D. Haines, Jr., MD's article entitled "The Ubiquitous Pharmaceutical Representative" [JOSMA, Dec 86] I became very upset by the one-sidedness of this article and felt obliged to respond. When Dr Haines feels that there should be a nationwide forum to decide what should be done with the professional pharmaceutical representative, one has to think should there be a nationwide forum on what to do with car salesmen, insurance salesmen, bankers, etc. The sale of pharmaceuticals is a business just like any other

(continued on p 180)

## Confer's experience (continued)

business. This business is under the supervision of hopefully knowledgeable and attentive physicians. The pharmaceutical representatives are a source of information for these supervisors. The problem is that the information base is constantly changing, either with new products or with new information about older products.

Professional pharmaceutical representatives, particularly those from large drug companies, are well informed about their products. If the physician asks the appropriate questions, he will get that information in a minimal amount of time. One problem is the very small amount of training students receive in their first two years of medical school regarding prescribing medications. As a graduate pharmacist, I was amazed how few medications the third-year medical student could write down on a piece of paper with dosage and frequency of administration. True, the concepts had been driven home, but for many I doubt that that large cloud of prescribing information ever descended upon them. So, to be safe, (and I agree with this concept) the young physician learns a couple of medications in each pharmaceutical category and looks up the others as needed. On the other hand, we may have a graduate pharmacist ("drug rep") who not only understands his own product, but most of his competitors; and, if he is good, he can give the drawbacks of his product as well as the advantages. But his wealth of information may be a bit overwhelming to a younger physician. The physician should also inquire about the cost and compare it to the competition. It is a service they provide and we should demand it.

My experience with professional pharmaceutical representatives has been exactly the opposite of Dr Haines. They have donated medications for indigent patients (our Tulsa free clinics are stocked with their sample medications); they have sponsored meetings and speakers that we could not otherwise have afforded; they continue to provide excellent surgical video tapes and cancer management tapes; and computer literature review service is available under their auspices (not to mention the traditional medical provisions to physicians and their families).

Regarding Dr Haines' article, I am not a "lazy physician" (except regarding yard work), yet I glean one or two useful pieces of information from drug representatives each month. As far as "wasted" office time, this can be a problem. My father was a career salesperson and he advised me to always take time to listen to what the salesman has to say. I do this, but never for more than ten minutes, always on a scheduled basis (unless they are from out of state), and no more frequently than every couple of months for each salesperson.

I think the "lazy doctor" being preyed upon by the "drug rep" is a bit overstated in Dr Haines' article. He sounds very busy, but hopefully someday he will have time to reopen his door to what can be a very useful source of information.

*David J. Confer, MD*
*Tulsa*


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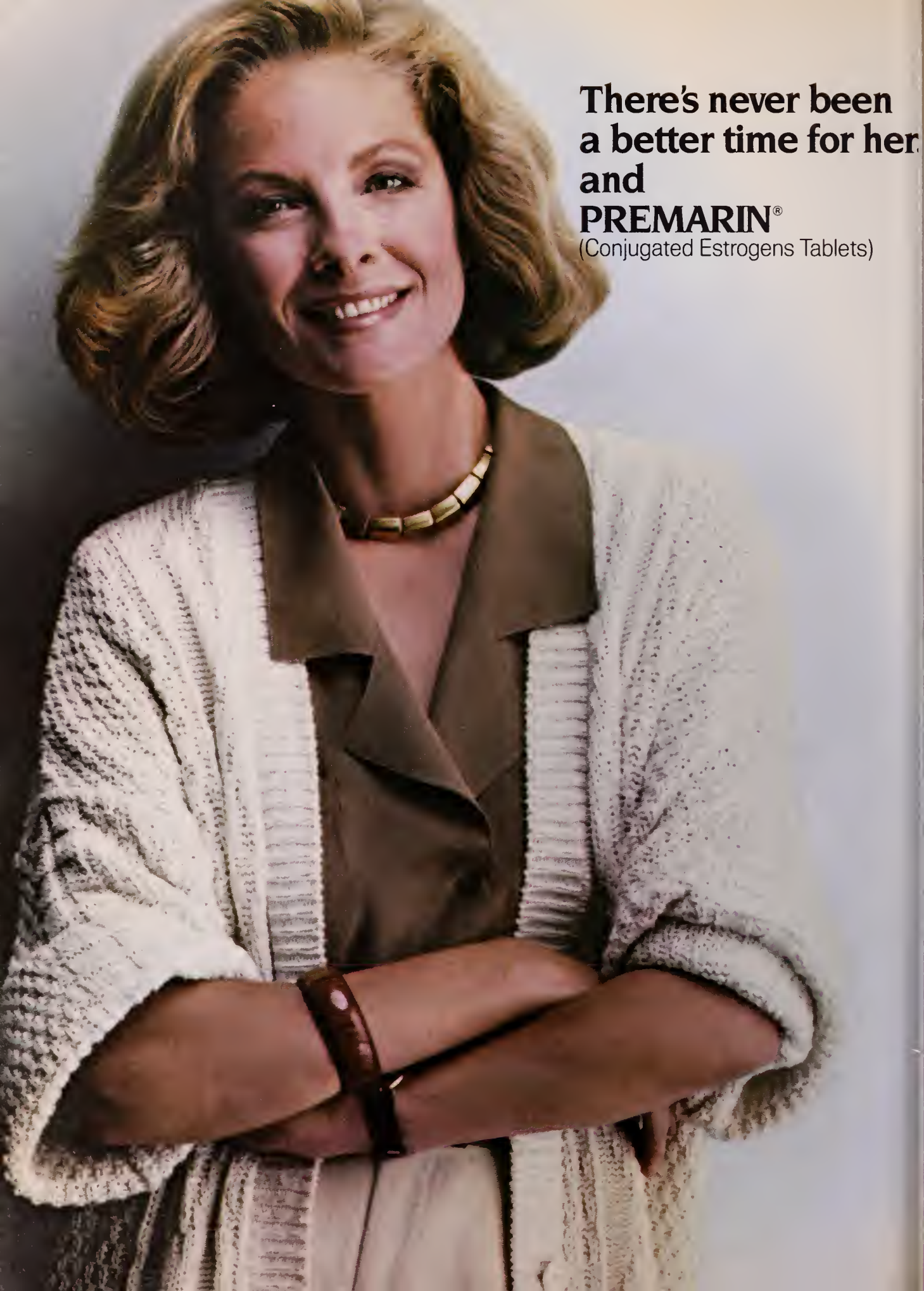
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a better time for her
and
PREMARIN®
(Conjugated Estrogens Tablets)

# Now the evidence looks better than ever

## Significantly reduced risk of endometrial hyperplasia

Endometrial hyperplasia was significantly reduced when progestin was added to PREMARIN therapy for more than ten days a month.[1-4] The risk of endometrial hyperplasia may also be reduced through cyclic administration of unopposed, low-dose PREMARIN.

## Effect on lipids—an important feature

PREMARIN used alone does not adversely affect lipid levels. In fact, a clinical study has shown a significant increase in HDL cholesterol—from 49.7 mg/dL to 56.4 mg/dL—and decrease in LDL cholesterol—from 165.1 mg/dL to 138.1 mg/dL—after one year of therapy with PREMARIN, 0.625 mg.[5]

## Low-dose control of menopausal symptoms*

PREMARIN effectively relieves vasomotor symptoms, such as hot flashes. When estrogen deficiency is limited to atrophic vaginitis, PREMARIN® (conjugated estrogens) Vaginal Cream restores the vaginal environment to its premenopausal state.

## The most widely used, most extensively studied estrogen worldwide.

# PREMARIN®

(Conjugated Estrogens Tablets)

## Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

# For moderate-to-severe vasomotor symptoms

# PREMARIN®
## (Conjugated Estrogens Tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

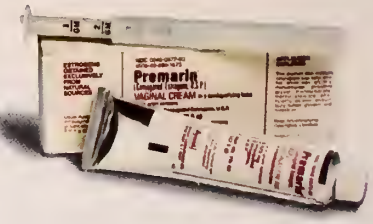
The appearance of these tablets is a trademark of Ayerst Laboratories.

# For atrophic vaginitis

# PREMARIN®
## (Conjugated Estrogens)

Vaginal Cream

0.625mg/g




BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

**PREMARIN® Brand of conjugated estrogens tablets, USP**
**PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base**

1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.

Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy. Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses.
2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY
The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring. It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4.7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

**DESCRIPTION:** PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.

**INDICATIONS AND USAGE:** PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass).** Atrophic vaginitis. Kraurosis vulvae. Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae. PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

**Concomitant Progestin Use:** The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS.) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.

**CONTRAINDICATIONS:** Estrogens should not be used in women (or men) with any of the following conditions: 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease. 2. Known or suspected estrogen-dependent neoplasia. 3. Known or suspected pregnancy (See Boxed Warning). 4. Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

**WARNINGS:** Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning.) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility. There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease, or abnormal mammograms. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk.

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed. Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.

**PRECAUTIONS:** Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Preexisting uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention.
b. Increased prothrombin and factors VII, VIII, IX, and X; decreased antithrombin 3; increased norepinephrine-induced platelet aggregability.
c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T4 by column, or T4 by radioimmunoassay. Free T3 resin uptake is decreased, reflecting the elevated TBG; free T4 concentration is unaltered.
d. Impaired glucose tolerance.
e. Decreased pregnanediol excretion.
f. Reduced response to metyrapone test.
g. Reduced serum folate concentration.
h. Increased serum triglyceride and phospholipid concentration. As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.

**ADVERSE REACTIONS:** The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea; premenstrual-like syndrome; amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine, dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance; aggravation of porphyria; edema; changes in libido.

**ACUTE OVERDOSAGE:** May cause nausea, and withdrawal bleeding may occur in females.

**DOSAGE AND ADMINISTRATION:**

**PREMARIN® Brand of conjugated estrogens tablets, USP**

1. *Given cyclically for short-term use only.* For treatment of moderate to severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.
2. *Given cyclically:* Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control. Osteoporosis—0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off).

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

**PREMARIN® Brand of conjugated estrogens Vaginal Cream**

*Given cyclically for short-term use only.* For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

**References:**

**1.** Whitehead MI, Townsend PT, Pryse-Davies J, et al: Effects of estrogens and progestins on the biochemistry and morphology of the postmenopausal endometrium. *N Engl J Med* 1981;305:1599-1605 **2.** Paterson MEL, Wade-Evans T, Sturdee DW, et al: Endometrial disease after treatment with oestrogens and progestogens in the climacteric. *Br Med J* 1980;280:822-824. **3.** Magos AL, Brincat M, Studd JWW, et al: Amenorrhea and endometrial atrophy with continuous oral estrogen and progestogen therapy in postmenopausal women. *Obstet Gynecol* 1985; 67: 496-499. **4.** Whitehead MI, Lane G, Siddle N, et al: Avoidance of endometrial hyperstimulation in estrogen-treated postmenopausal women. *Semin Reprod Endocrinol* 1983;1:1;41-52. **5.** Barnes RB, Roy S, Lobo RA: Comparison of lipid and androgen levels after conjugated estrogen or depo-medroxyprogesterone acetate treatment in postmenopausal women. *Obstet Gynecol* 1985;66:216-219.

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ZANTAC® 150 h.s.

ranitidine HCl/Glaxo 150 mg tablets

EFFECTIVE MAINTENANCE THERAPY

for healed duodenal ulcer patients

CONFIRMED

In two randomized, double-blind, and well-controlled clinical trials, ZANTAC 150 mg h.s. significantly superior to cimetidine 400 mg h.s. for maintenance therapy in healed duodenal ulcers.

Percent of patients with observed duodenal ulcer recurrence

| | | 0-4 months | 0-8 months | 0-12 months | No. patients |
|---|---|---|---|---|---|
| USA[1] | ranitidine 150 mg h.s. | 9% | 14%* | 16%† | 60 |
| | cimetidine 400 mg h.s. | 23% | 34% | 43% | 66 |
| UK, Ireland, Australia[2] | ranitidine 150 mg h.s. | 8%‡ | 14%‡ | 23%‡ | 243 |
| | cimetidine 400 mg h.s. | 21% | 34% | 37% | 241 |

*p=0.02
†p=0.01
‡p≤0.004
%=life-table estimates
All patients were permitted prn antacids for relief of pain.

These two trials used the currently recommended dosing regimen of cimetidine (400 mg h.s.) and ranitidine (150 mg h.s.). A comparison of other dosing regimens has not been studied.

The studied dosing regimens are not equivalent with respect to the degree and duration of acid suppression or suppression of nocturnal acid.

The superiority of ranitidine over cimetidine in these trials indicates that the dosing regimen currently recommended for cimetidine is less likely to be as successful in maintenance therapy.

Convenient once-a-night dose with a

low incidence of side effects[3]

Headache, sometimes severe, seems to be related to ranitidine administration. Other side effects have been reported; for a complete listing, see the ADVERSE REACTIONS section in the Brief Summary.

No significant interference with the hepatic cytochrome

P-450 enzyme system at recommended doses

ZANTAC 150 mg has no significant drug interactions with theophylline, phenytoin, or warfarin. The bioavailability of certain medications whose absorption is dependent on a low gastric pH may be altered when ZANTAC or other medications that decrease gastric acidity are administered.

**Zantac® 150**

*ranitidine HCl/Glaxo 150 mg tablets*

One tablet at bedtime
for maintenance

See next page for references and
Brief Summary of Product Information.

Glaxo/ROCHE®

CONFIRMED

# Zantac 150

ranitidine HCl/Glaxo 150 mg tablets

## *One tablet at bedtime for maintenance therapy in healed duodenal ulcer patients*


**References:**

1. Silvis SE, Griffin J, Hardin R, et al: Final report on the United States multicenter trial comparing ranitidine to cimetidine as maintenance therapy following healing of duodenal ulcer. *J Clin Gastroenterol* 1985;7(6):482-487.
2. Gough KR, Korman MG, Bardhan KD, et al: Ranitidine and cimetidine in prevention of duodenal ulcer relapse: A double-blind, randomised, multicentre, comparative trial. *Lancet* 1984;ii:659-662.
3. Data available on request, Glaxo Inc.


**ZANTAC® 150 Tablets (ranitidine hydrochloride)**
**ZANTAC® 300 Tablets (ranitidine hydrochloride)**

**BRIEF SUMMARY OF PRODUCT INFORMATION**

The following is a brief summary only. Before prescribing, see complete prescribing information in ZANTAC® product labeling.

**INDICATIONS AND USAGE:** ZANTAC® is indicated in:

1. Short-term treatment of **active duodenal ulcer.** Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD)**. Symptomatic relief commonly occurs within one or two weeks after starting therapy and is maintained throughout a six-week course of therapy.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

**CONTRAINDICATIONS:** ZANTAC® is contraindicated for patients known to have hypersensitivity to the drug.

**PRECAUTIONS:** Symptomatic response to ZANTAC® therapy does not preclude the presence of gastric malignancy.

Since ZANTAC is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see **DOSAGE AND ADMINISTRATION**). Caution should be observed in patients with hepatic dysfunction since ZANTAC is metabolized in the liver.

False-positive tests for urine protein with Multistix® may occur during ZANTAC therapy, and therefore testing with sulfosalicylic acid is recommended.

Although recommended doses of ZANTAC do not inhibit the action of cytochrome P-450 enzymes in the liver, there have been isolated reports of drug interactions which suggest that ZANTAC may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Lack of experience to date precludes recommending ZANTAC for use in children or pregnant patients. Since ZANTAC is secreted in human milk, caution should be exercised when administered to a nursing mother.

**ADVERSE REACTIONS:** Headache, sometimes severe, seems to be related to ZANTAC® administration. Constipation, diarrhea, nausea/vomiting, and abdominal discomfort/pain have been reported. There have been rare reports of malaise, dizziness, somnolence, insomnia, vertigo, tachycardia, bradycardia, premature ventricular beats, and arthralgias. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients.

In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid IV for seven days, and in 4 of 24 subjects receiving 50 mg qid for five days. With oral administration there have been occasional reports of reversible hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice.

There have been rare reports of reversible leukopenia, granulocytopenia, thrombocytopenia, and pancytopenia.

Although controlled studies have shown no antiandrogenic activity, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving ZANTAC, but the incidence did not differ from that in the general population.

Incidents of rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia, have been reported, as well as rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia) and small increases in serum creatinine.

**OVERDOSAGE:** Information concerning possible overdosage and its treatment appears in the full prescribing information.

**DOSAGE AND ADMINISTRATION. Active Duodenal Ulcer:** The current recommended adult oral dosage is 150 mg twice daily. An alternate dosage of 300 mg once daily at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated.

**Maintenance Therapy:** The current recommended adult oral dosage is 150 mg at bedtime.

**Pathological Hypersecretory Conditions (such as Zollinger-Ellison Syndrome):** The current recommended adult oral dosage is 150 mg twice a day. In some patients it may be necessary to administer ZANTAC 150-mg doses more frequently. Doses should be adjusted to individual patient needs, and should continue as long as clinically indicated. Doses up to 6 g/day have been employed in patients with severe disease.

**Benign Gastric Ulcer:** The current recommended adult oral dosage is 150 mg twice a day.

**GERD:** The current recommended adult oral dosage is 150 mg twice a day.

**Dosage Adjustment for Patients with Impaired Renal Function:** On the basis of experience with a group of subjects with severely impaired renal function treated with ZANTAC, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

**HOW SUPPLIED:** ZANTAC® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) and unit dose packs of 100 tablets (NDC 0173-0393-47).

ZANTAC® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 tablets (NDC 0173-0344-42) and unit dose packs of 100 tablets (NDC 0173-0344-47).

**Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.**

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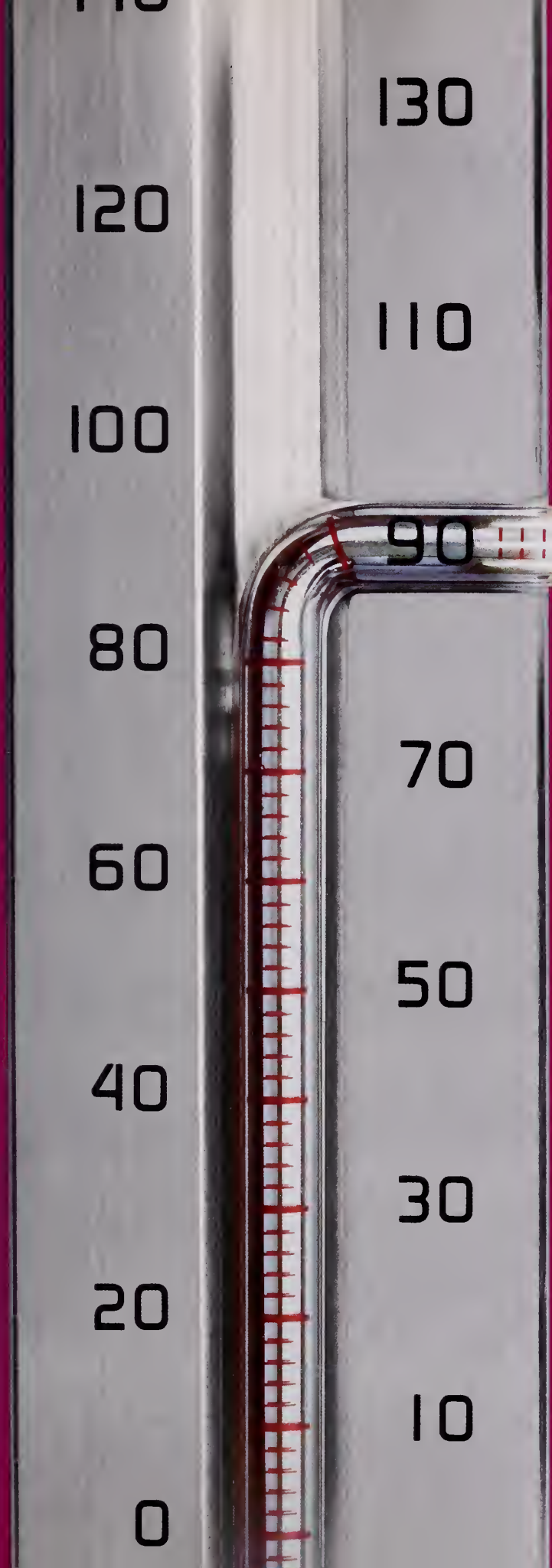


In mild to moderate hypertension

# THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER

**NEW**
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# ISOPTIN SR*
## (verapamil HCl/Knoll)

*240 mg scored, sustained-release tablets*

**JAMES B.**
38, black male, heavy smoker. Prescribed a diuretic by another physician last year for hypertension.

**YOUR CONCERNS**
Presents with "smoker's cough." Workup reveals a BP of 150/107.

**A LOGICAL CHOICE FOR CONTROL OF HIS BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Black hypertensives often have low plasma renin activity and generally do not respond favorably to beta blockers.
— Beta blockers may increase the likelihood of bronchospasm.

**ALICE W.**
65, diabetic, overweight. Her BP has elevated to 190/98.

**YOUR CONCERNS**
She's on daily insulin.

**A LOGICAL CHOICE FOR CONTROL OF HER BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Unlike most beta blockers and diuretics, ISOPTIN has no adverse effects on serum glucose levels.
— Unlike most beta blockers, ISOPTIN does not mask the symptoms of hypoglycemia.

**THOMAS G.**
70, asthmatic. In the past, BP adequately controlled with 25 mg hydrochlorothiazide daily.

**YOUR CONCERNS**
Today patient presents with symptoms of gout. Workup reveals high uric acid level, low serum potassium, and BP elevated to 180/98.

**A LOGICAL CHOICE FOR CONTROL OF HIS BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Unlike diuretics, ISOPTIN will not decrease serum potassium levels or elevate uric acid levels.
— Unlike beta blockers, ISOPTIN can be used safely in asthma and COPD patients.

**JOHN K.**
42, Annual physical uncovered diastolic BP of 102... confirmed on three successive office visits. Unresponsive to nonpharmacologic intervention.

**YOUR CONCERNS**
Salesman, spends many hours of his working day in car... total cholesterol level 300, HDL 35.

**A LOGICAL CHOICE FOR CONTROL OF HIS BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Unlike diuretics, ISOPTIN does not cause urinary urgency.
— Unlike either beta blockers or diuretics, ISOPTIN will not adversely affect his already seriously compromised lipid profile.
— Unlike with propranolol, fatigue and impotence are rarely reported.

## *Antihypertensive therapy you and your patients can live with*

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*In mild to moderate hypertension* Brief Summary

*THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER*

**ISOPTIN® SR (verapamil HCl/Knoll) 240 mg scored, sustained-release tablets**

**CONTRAINDICATIONS:** 1) Severe left ventricular dysfunction (see WARNINGS), 2) Hypotension less than 90 mmHg systolic pressure) or cardiogenic shock, 3) Sick sinus syndrome or 2nd or 3rd degree AV block (except in patients with a functioning artificial ventricular pacemaker).

**WARNINGS: Heart Failure:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (see DRUG INTERACTIONS). Patients with milder ventricular dysfunction should, if possible, be controlled before verapamil treatment. Hypotension: ISOPTIN (verapamil HCl) may produce occasional symptomatic hypotension. Elevated Liver Enzymes: Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Periodic monitoring of liver function in patients receiving verapamil is therefore prudent. Accessory Bypass Tract (Wolff-Parkinson-White): Patients with paroxysmal and/or chronic atrial flutter or atrial fibrillation and a coexisting accessory AV pathway have developed increased antegrade conduction across the accessory pathway producing a very rapid ventricular response or ventricular fibrillation after receiving intravenous verapamil. While this has not been reported with oral verapamil, it should be considered a potential risk. Treatment is usually D.C.-cardioversion. Atrioventricular Block: The effect of verapamil on AV conduction and the SA node may cause asymptomatic 1st degree AV block and transient bradycardia. Higher degrees of AV block, while infrequent (0.8%), may require a reduction in dosage or, in rare instances, discontinuation of verapamil HCl. Patients with Hypertrophic Cardiomyopathy (IHSS): Although verapamil has been used in the therapy of patients with IHSS, severe cardiovascular decompensation and death have been noted in this patient population.

**PRECAUTIONS: Impaired Hepatic or Renal Function:** Verapamil is highly metabolized by the liver with about 70% of an administered dose excreted in the urine. In patients with impaired hepatic or renal function verapamil should be administered cautiously and the patients monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacological effects (see OVERDOSAGE).

**Drug Interactions:** Beta Blockers: Concomitant use of ISOPTIN and oral beta-adrenergic blocking agents may be beneficial in certain patients with chronic stable angina or hypertension, but available information is not sufficient to predict with confidence the effects of concurrent treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities. Digitalis: Clinical use of verapamil in digitalized patients has shown the combination to be well tolerated if digoxin doses are properly adjusted. However, chronic verapamil treatment increases serum digoxin levels by 50 to 75% during the first week of therapy and this can result in digitalis toxicity. Upon discontinuation of ISOPTIN (verapamil HCl), the patient should be reassessed to avoid underdigitalization. Antihypertensive Agents: Verapamil administered concomitantly with oral antihypertensive agents (e.g., vasodilators, angiotensin-converting enzyme inhibitors, diuretics, beta blockers, prazosin) will usually have an additive effect on lowering blood pressure. Patients receiving these combinations should be appropriately monitored. Disopyramide: Disopyramide should not be administered within 48 hours before or 24 hours after verapamil administration. Quinidine: In patients with hypertrophic cardiomyopathy (IHSS), concomitant use of verapamil and quinidine resulted in significant hypotension. There has been a report of increased quinidine levels during verapamil therapy. Nitrates: The pharmacologic profile of verapamil and nitrates as well as clinical experience suggest beneficial interactions. Cimetidine: Two clinical trials have shown a lack of significant verapamil interaction with cimetidine. A third study showed cimetidine reduced verapamil clearance and increased elimination to 1/2. Anesthetic Agents: Verapamil may potentiate the activity of neuromuscular blocking agents and inhalation anesthetics. Carbamazepine: Verapamil may increase carbamazepine concentrations during combined therapy. Rifampin: Therapy with rifampin may markedly reduce oral verapamil bioavailability. Lithium: Verapamil may lower lithium levels in patient on chronic oral lithium therapy. Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no evidence of a carcinogenic potential of verapamil administered to rats for two years. Verapamil was not mutagenic in the Ames test. Studies in female rats did not show impaired fertility. Effects on male fertility have not been determined. Pregnancy (Category C): There are no adequate and well-controlled studies in pregnant women. ISOPTIN crosses the placental barrier and can be detected in umbilical vein blood at delivery. This drug should be used during pregnancy, labor, and delivery, only if clearly needed. Nursing Mothers: ISOPTIN is excreted in human milk, therefore, nursing should be discontinued while verapamil is administered. Pediatric Use: Safety and efficacy of ISOPTIN in children below the age of 18 years have not been established.

**ADVERSE REACTIONS:** Constipation 8.4%, dizziness 3.5%, nausea 2.7%, hypotension 2.5%, edema 2.1%, headache 1.9%, CHF/pulmonary edema 1.8%, fatigue 1.7%, bradycardia 1.4%, 1° AV block 0.8%, flushing 0.1%, elevated liver enzymes (see WARNINGS). The following reactions, reported in less than 1.0% of patients, occurred under conditions (open trials, marketing experience) where a causal relationship is uncertain; they are mentioned to alert the physician to a possible relationship: angina pectoris, arthralgia and rash, AV block, blurred vision, cerebrovascular accident, chest pain, claudication, confusion, diarrhea, dry mouth, dyspnea, ecchymosis or bruising, equilibrium disorders, exanthema, gastrointestinal distress, gingival hyperplasia, gynecomastia, hair loss, hyperkeratosis, impotence, increased urination, insomnia, macules, muscle cramps, myocardial infarction, palpitations, paresthesia, psychotic symptoms, purpura (vasculitis), shakiness, somnolence, spotty menstruation, sweating, syncope, urticaria. Treatment of Acute Cardiovascular Adverse Reactions: Whenever severe hypotension or complete AV block occur following oral administration of verapamil, the appropriate emergency measures should be applied immediately, e.g., intravenously administered isoproterenol HCl, levarterenol bitartrate, atropine (all in the usual doses), or calcium gluconate (10% solution). If further support is necessary, inotropic agents (dopamine or dobutamine) may be administered. Actual treatment and dosage should depend on the severity and the clinical situation and the judgment and experience of the treating physician.

**OVERDOSAGE:** Treatment of overdosage should be supportive. Beta-adrenergic stimulation or parenteral administration of calcium solutions may increase calcium ion flux across the slow channel, and have been used effectively in treatment of deliberate overdosage with verapamil. Clinically significant hypotensive reactions or fixed high degree AV block should be treated with vasopressor agents or cardiac pacing, respectively. Asystole should be handled by the usual measures including cardiopulmonary resuscitation.

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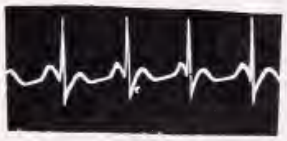

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# Doctor's Day

On March 30, local organizations around the nation will observe Doctor's Day! This is the day where we honor physicians for their dedication to the practice of medicine. It gives all auxiliaries and communities the chance to tell doctors how much we appreciate all their hard work and all the time they have given to helping mankind. With the face of medicine changing all the time (such as outside influences like government involvement), it is especially important for us to pay tribute to the men and women who are our doctors.

In order to understand Doctor's Day and how it originated, a brief history of Doctor's Day should be mentioned. Mrs Eudora Brown Almond was brought up in a small community and was greatly impressed by her family physician. In 1920, she married Dr Charles B. Almond and recognized his dedication to the practice of medicine. She decided that something special should be done for doctors in her county, which was Barrow County in Georgia. At the suggestion of Mrs Almond, Georgia adopted a resolution to pay tribute to its doctors. The first celebration of Doctor's Day was on March 30, 1933, by the Barrow County auxiliary. March 30 was chosen as the official day for Doctor's Day by the Southern Medical Association Auxiliary in 1935 to honor Dr Crawford Long of Georgia who, on March 30, 1842, first used ether as an anesthetic.

There are several ways in which to observe Doctor's Day. Different auxiliaries around the state of Oklahoma have come up with some ingenious ways of celebrating it. Some have had dinners and members came dressed as they were in high school. One auxiliary had a health fair, another co-hosted a blood drive, and others have hosted gourmet dinners. Still others have donated money to hospitals or health-care related fields.

The symbol of Doctor's Day is the red carnation. Many auxiliaries around the state provide their doctors with carnations that they wear proudly on their lapel on the 30th. This is one way of showing the doctors that this is their special day and they are appreciated. Red carnations could also be put in hospitals, churches, offices, on graves of deceased physicians, or at the Doctor's Day dinner that evening.

Let's make Doctor's Day 1987 a meaningful and special occasion. Physicians work long and hard hours. They care about their patients. We need to let physicians know how much we value their dedication to the practice of medicine. Remember to offer even just a simple "thank you" when you see any physician that you know, to let him or her know that they are appreciated and that we know they are practicing medicine to benefit mankind.

■ **The Oklahoma State Medical Association** would like to take this opportunity to thank Whitehall Laboratories and Jackie Steinbauer, in particular, for their generosity in supporting the OSMA's first aid station at the state capitol. Their provision of assorted over-the-counter medications for the volunteer first aid station is very much appreciated.

■ **Robert C. Bowman, MD, Nowata, and Lee N.** Newcomer, MD, Tulsa, have been selected to represent Oklahoma in the Young Physician Section of the AMA House of Delegates. Dr Bowman, a family physician, is the state delegate, and Dr Newcomer, an oncologist, will serve as alternate delegate. The newly created section, which will address the concerns of physicians under forty years of age or in practice for less than five years, held its first meeting in December.

■ **Project USA, an AMA program, is recruiting** fully licensed physicians for short-term, general medicine assignments at Indian Health Service (IHS) and National Health Service Corps (NHSC) hospitals and clinics. The majority of vacancies are for two weeks, with some extending to four weeks. Participating physicians receive $750 a week at IHS facilities and $500 a week at NHSC clinics. Doctors are reimbursed for round trip transportation expenses, and living accommodations are provided. Interested physicians should contact John Naughton, project director, at the AMA, 535 North Dearborn, Chicago, IL 60610, or call (312) 645-4702.

■ **Under the headline "Assignment law should** spark new revolution," *American Medical News* has published a lengthy letter from Ray V. McIntyre, MD, Kingfisher. In the letter, appearing in the My Opinion column of the February 6 issue, Dr McIntyre expresses his deep concern about the new Massachusetts law requiring doctors to accept Medicare assignments or lose their licenses to practice. "To accomplish this questionable purpose," he says, "the state will pervert the licensing process by requiring an involuntary contract with a federal agency of the United States government. As an assignment is a contract that is unilaterally written, unconditional, lasts forever, and non-negotiable, it is essentially involuntary servitude. Sadly, three or four other states are considering similar laws."

■ **"Clinical Problems in Ophthalmology"** and "Current Trends in Pediatric Therapy XIII" are the CME programs being offered by the University of Oklahoma College of Medicine in April. The Saturday, April 11, ophthalmology program will review the current management of clinical problems in the field, emphasizing the new developments. The April 23-25 (Thursday-Saturday) pediatrics program is designed to update pediatricians and generalists in the application of current medical thought regarding changing concepts of treatment. The program, to be conducted at Fountainhead Resort, Checotah, is designed to meet the needs of the pediatrician and the generalist who deals with children. There is no charge for these programs. Doctors are urged to confirm their plans to attend by contacting Magdalen De Bault, associate director, CME, OU College of Medicine, PO Box 26901, Room 164E, LB, Oklahoma City, OK 73190.

■ **The AMA conference "DNA Probes in the** Practice of Medicine" will be Saturday and Sunday, April 25 and 26, at the Hyatt Regency Washington Hotel in Washington, DC. The program will examine the state-of-the-art medical uses of DNA probes. For additional information on the conference, contact R. Mark Evans, PhD, Office of Biotechnology, AMA, 535 North Dearborn, Chicago, IL 60610, (312) 645-4567.

■ **Oklahomans Against Lawsuit Abuse (OALA)** and the Oklahoma State Chamber of Commerce (OCCI) have joined forces to restore "balanced civil justice" through reform of the state's existing tort laws. Brochures explaining Senate Bill 134 on tort reform are now being distributed. Additional brochures are available through local chambers of commerce or by contacting OALA, OCCI, or the OSMA. OSMA members are being asked to give the brochures to friends and associates and to seek their immediate support in the drive for tort reform.

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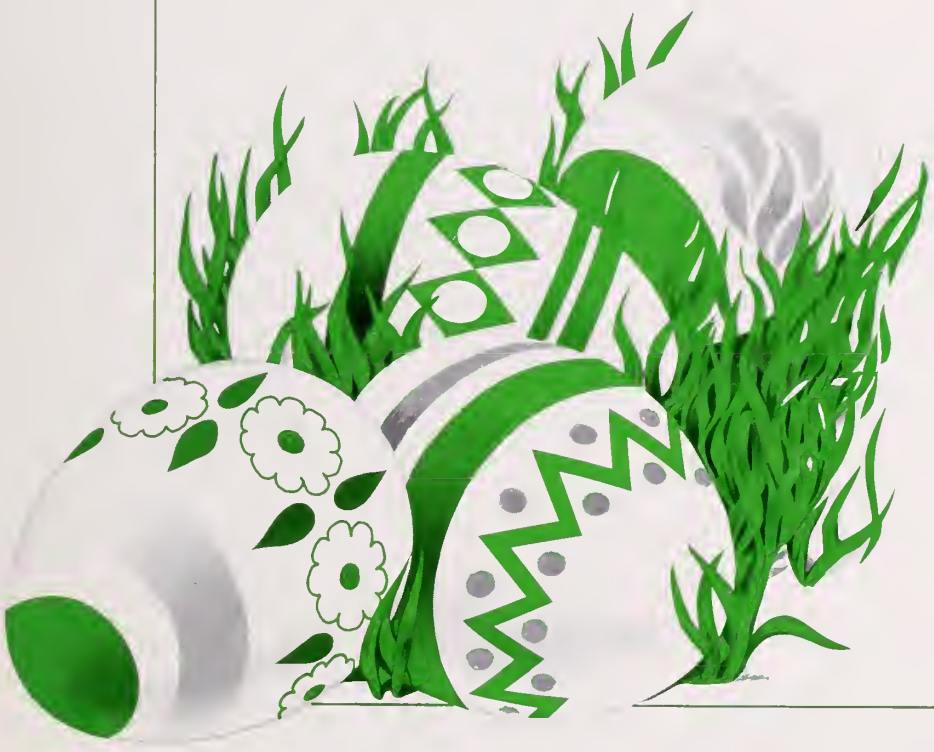
APRIL 1987



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# JOURNAL

## OKLAHOMA STATE MEDICAL ASSOCIATION

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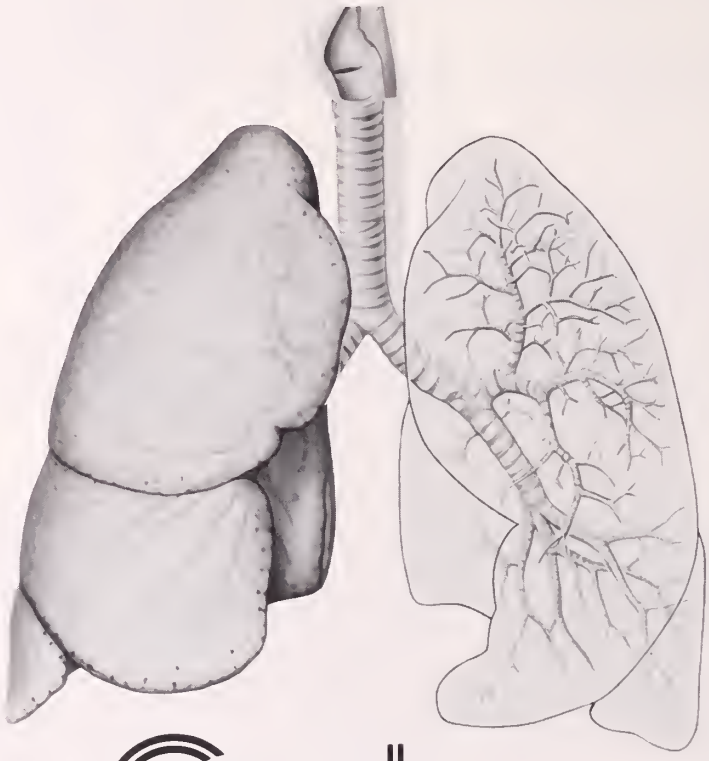

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**Contraindications:** Known allergy to cephalosporins.

**Warnings:** CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

**Precautions:**
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- In renal impairment, safe dosage of Ceclor may be lower than that usually recommended. Ceclor should be administered with caution in such patients.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

**Adverse Reactions:** (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, erythema multiforme, serum-sickness-like reactions): 1.5%; usually subside within a few days after cessation of therapy. These reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%.

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*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

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Please see next page for brief summary of prescribing information.

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**DESCRIPTION.** Inderal LA is formulated to provide a sustained release of propranolol hydrochloride. Inderal LA is available as 80 mg, 120 mg, and 160 mg capsules.

**CLINICAL PHARMACOLOGY.** INDERAL is a nonselective beta-adrenergic receptor blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg for mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

The mechanism of the antihypertensive effect of INDERAL has not been established. Among the factors that may be involved in contributing to the antihypertensive action are (1) decreased cardiac output, (2) inhibition of renin release by the kidneys, and (3) diminution of tonic sympathetic nerve outflow from vasomotor centers in the brain. Although total peripheral resistance may increase initially, it readjusts to or below the pretreatment level with chronic use. Effects on plasma volume appear to be minor and somewhat variable. INDERAL has been shown to cause a small increase in serum potassium concentration when used in the treatment of hypertensive patients.

In angina pectoris, propranolol generally reduces the oxygen requirement of the heart at any given level of effort by blocking the catecholamine-induced increases in the heart rate, systolic blood pressure, and the velocity and extent of myocardial contraction. Propranolol may increase oxygen requirements by increasing left ventricular fiber length, end diastolic pressure and systolic ejection period. The net physiologic effect of beta-adrenergic blockade is usually advantageous and is manifested during exercise by delayed onset of pain and increased work capacity.

In dosages greater than required for beta blockade, INDERAL also exerts a quinidine-like or anesthetic-like membrane action which affects the cardiac action potential. The significance of the membrane action in the treatment of arrhythmias is uncertain.

The mechanism of the antimigraine effect of propranolol has not been established. Beta-adrenergic receptors have been demonstrated in the pial vessels of the brain.

Beta receptor blockade can be useful in conditions in which, because of pathologic or functional changes, sympathetic activity is detrimental to the patient. But there are also situations in which sympathetic stimulation is vital. For example, in patients with severely damaged hearts, adequate ventricular function is maintained by virtue of sympathetic drive which should be preserved. In the presence of AV block, greater than first degree, beta blockade may prevent the necessary facilitating effect of sympathetic activity on conduction. Beta blockade results in bronchial constriction by interfering with adrenergic bronchodilator activity which should be preserved in patients subject to bronchospasm.

Propranolol is not significantly dialyzable.

**INDICATIONS AND USAGE. Hypertension:** INDERAL LA is indicated in the management of hypertension, it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

**Angina Pectoris Due to Coronary Atherosclerosis:** INDERAL LA is indicated for the long-term management of patients with angina pectoris.

**Migraine:** INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

**Hypertrophic Subaortic Stenosis:** INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

**CONTRAINDICATIONS.** INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

**WARNINGS.** CARDIAC FAILURE. Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually if possible).

IN PATIENTS WITH ANGINA PECTORIS there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following abrupt discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

**Nonallergic Bronchospasm (e.g., chronic bronchitis, emphysema)** — PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY. The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, e.g., dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA. Beta-adrenergic blockade may prevent the appearance of certain premonitory signs and symptoms (pulse rate and pressure changes) of acute hypoglycemia in labile insulin-dependent diabetes. In these patients, it may be more difficult to adjust the dosage of insulin.

THYROTOXICOSIS. Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol does not distort thyroid function tests.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranolol.

**PRECAUTIONS.** *General.* Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

*Clinical Laboratory Tests.* Elevated blood urea levels in patients with severe heart disease; elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS. Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

*Carcinogenesis, Mutagenesis, Impairment of Fertility.* Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

*Pregnancy.* Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

*Nursing Mothers.* INDERAL is excreted in human milk. Caution should be exercised when INDERAL is administered to a nursing woman.

*Pediatric Use.* Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS.** Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

*Cardiovascular.* bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency usually of the Raynaud type.

*Central Nervous System.* lightheadedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics.

*Gastrointestinal.* nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

*Allergic.* pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

*Respiratory.* bronchospasm.

*Hematologic.* agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

*Auto-Immune.* In extremely rare instances, systemic lupus erythematosus has been reported.

*Miscellaneous.* alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence, and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

**DOSAGE AND ADMINISTRATION.** INDERAL LA provides propranolol hydrochloride in a sustained-release capsule for administration once daily. If patients are switched from INDERAL tablets to INDERAL LA capsules, care should be taken to assure that the desired therapeutic effect is maintained. INDERAL LA should not be considered a simple mg for mg substitute for INDERAL. INDERAL LA has different kinetics and produces lower blood levels. Retitration may be necessary especially to maintain effectiveness at the end of the 24-hour dosing interval.

HYPERTENSION — *Dosage must be individualized.* The usual initial dosage is 80 mg INDERAL LA once daily, whether used alone or added to a diuretic. The dosage may be increased to 120 mg once daily or higher until adequate blood-pressure control is achieved. The usual maintenance dosage is 120 to 160 mg once daily. In some instances a dosage of 640 mg may be required. The time needed for full hypertensive response to a given dosage is variable and may range from a few days to several weeks.

ANGINA PECTORIS — *Dosage must be individualized.* Starting with 80 mg INDERAL LA once daily, dosage should be gradually increased at three to seven day intervals until optimum response is obtained. Although individual patients may respond at any dosage level, the average optimum dosage appears to be 160 mg once daily. In angina pectoris, the value and safety of dosage exceeding 320 mg per day have not been established.

If treatment is to be discontinued, reduce dosage gradually over a period of a few weeks (see WARNINGS).

MIGRAINE — *Dosage must be individualized.* The initial oral dose is 80 mg INDERAL LA once daily. The usual effective dose range is 160-240 mg once daily. The dosage may be increased gradually to achieve optimum migraine prophylaxis. If a satisfactory response is not obtained within four to six weeks after reaching the maximum dose, INDERAL LA therapy should be discontinued. It may be advisable to withdraw the drug gradually over a period of several weeks.

HYPERTROPHIC SUBAORTIC STENOSIS — 80-160 mg INDERAL LA once daily.

PEDIATRIC DOSAGE — At this time the data on the use of the drug in this age group are too limited to permit adequate directions for use.

*The appearance of these capsules is a registered trademark of Ayerst Laboratories.

**REFERENCES:**


1. INDERAL LA National Compliance Evaluation Program. Data on file. Ayerst Laboratories.
2. Ravid M, Lang R, Jutrin I. The relative antihypertensive potency of propranolol, oxprenolol, atenolol, and metoprolol given once daily. *Arch Intern Med* 1985; 145:1321-1323.


**6412/187**

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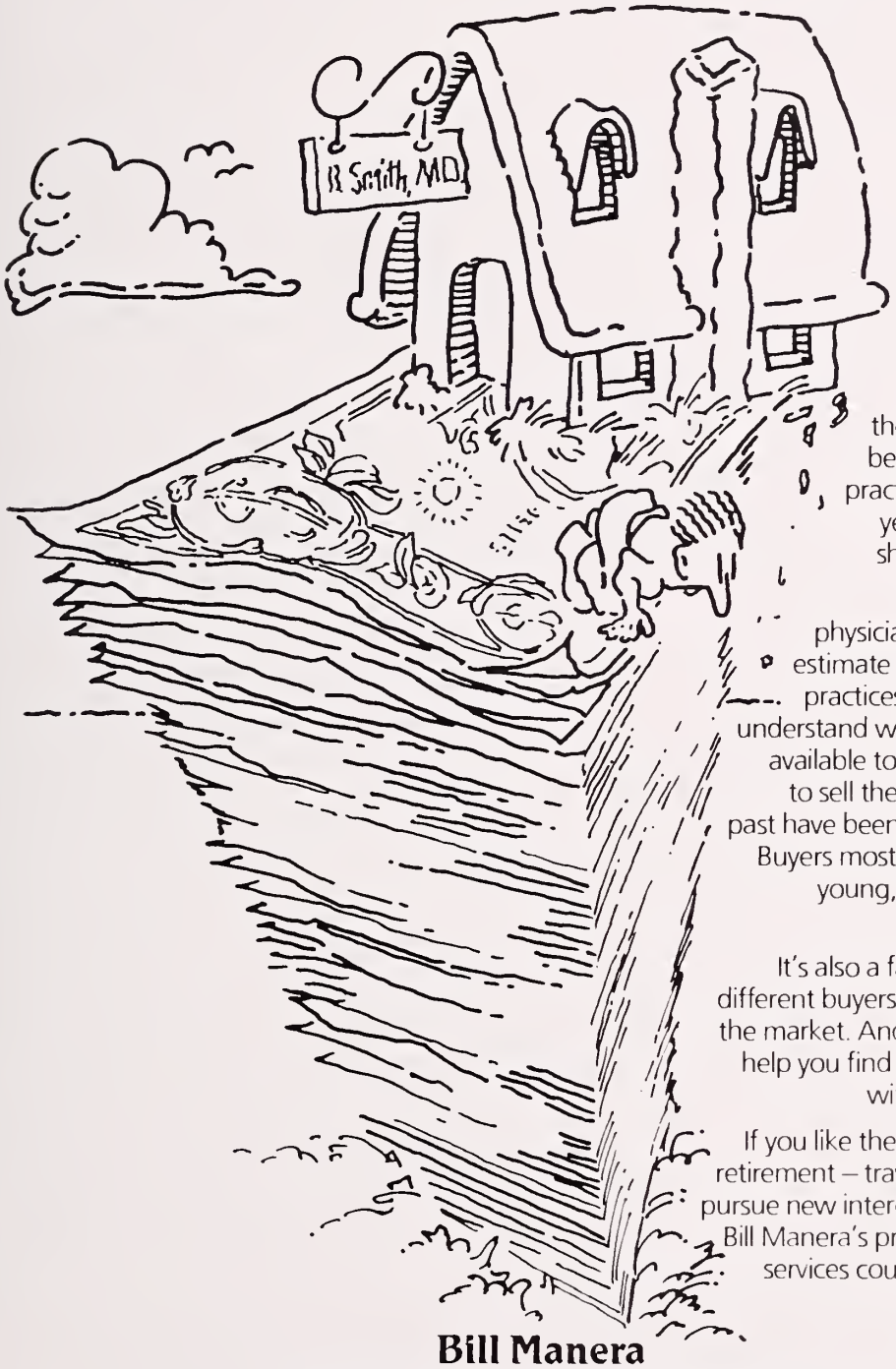
# Your practice may be worth much more than you thought.

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**Help us fight the odds.**

**Prehospital emergency care needs physician support. The Oklahoma EMT Association is a non-profit organization dedicated to prehospital medical excellence. For more information on how you can be involved in your local EMS, or to make a tax-deductible contribution, contact the OEMTA.**

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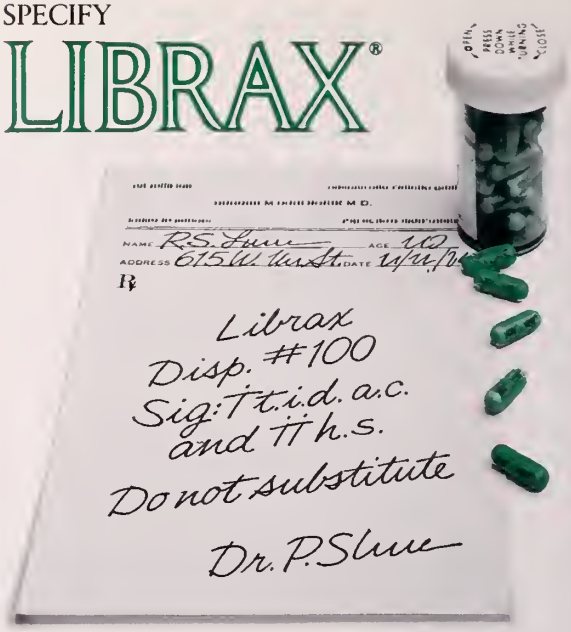
SPECIFY

# LIBRAX®




Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide

Please consult complete prescribing information, a summary of which follows:

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis. Final classification of the less-than-effective indications requires further investigation.

**Contraindications:** Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.

**Warnings:** Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

*Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.

**Precautions:** In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.

**Adverse Reactions:** No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

ROCHE®
Roche Products Inc.
Manati, Puerto Rico 00701

ROCHE

# FLARE-UP

## SPASM AND PAIN CAN SIGNAL FUNCTIONAL GI DISORDERS*

Patients experiencing symptoms of irritable bowel syndrome* or duodenal ulcer* can often have emotional stress operating in the background. When you prescribe Librax for these patients, they receive treatment for both the emotional and the somatic elements to help relieve the anxiety/pain cycle.

Librax provides the well-known antianxiety action of Librium® (chlordiazepoxide HCl/ Roche), a benzodiazepine with an established record of safety after use in thousands of patients worldwide. Also included are the proven antispasmodic and antisecretory actions of Quarzan® (clidinium bromide/ Roche), the component which helps to reduce colonic spasm and hypersecretion and helps also to alleviate the pain they cause.

## LIBRAX: FOR THE DUAL PROBLEMS OF FUNCTIONAL GI DISORDERS.

SPECIFY ADJUNCTIVE

# LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide.

ANTIANXIETY
ANTISECRETORY
ANTISPASMODIC

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of duodenal ulcer and the irritable bowel syndrome.

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Please see reverse side for complete product information.

# LOSS PREVENTION SEMINAR ATTENDANCE MANDATORY

Attendance at an OSMA-PLICO sponsored Loss Prevention Seminar is now mandatory for all PLICO-insured physicians at least once every three years. **If a physician has never attended a seminar, he or she must attend one during 1987.** If a physician has not attended a program since 1984, he or she must attend this year, also.

The change making seminar attendance mandatory was implemented by the PLICO Board of Directors in late 1985, and a special endorsement outlining the requirement was included in all 1986 PLICO professional liability insurance policies. Any physician needing to attend in 1987, and failing to do so, will not be eligible for renewal of his or her PLICO professional liability insurance for calendar year 1988.

A Seminar Registration Form is located at the bottom of this page and a schedule of upcoming seminars is included. The registration form may be used for any seminar, but please **specify the date** you would like to attend. It is advisable to pre-register for the seminar you would like to attend.

## OSMA-PLICO
## 1987 LOSS PREVENTION SEMINARS

| | | |
|---|---|---|
| May 1 | (in conjunction with OSMA Annual Meeting) Fri. 9-12 Noon | OKLAHOMA CITY Sheraton Century |
| June 6 | Sat. 2-5 p.m. | TULSA Marriott |
| July 18 | Sat. 2-5 p.m. | LAWTON Holiday Inn |
| August 8 | Sat. 2-5 p.m. | WOODWARD Park Inn |
| September 19 | Sat. 8:30 a.m.-11:30 a.m. | McALESTER Holiday Inn |
| September 26 | Sat. 8:30 a.m.-11:30 a.m. | OKLAHOMA CITY Lincoln Plaza |

## LOSS PREVENTION SEMINAR REGISTRATION

Please register the following physician to attend the 1987 Loss Prevention Seminar indicated below (PLEASE TYPE OR PRINT):

Physician's Name: _____

Address: _____ City/State _____ Zip _____

Seminar Date and City: _____

Mail form to Oklahoma State Medical Association, 601 NW Expressway, Oklahoma City, OK 73118.

"Living in the city is lonely enough... with herpes it's like solitary confinement."
ZOVIRAX®
(acyclovir)
CAPSULES
Prevent genital herpes recurrences month after month with daily therapy.
(In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.)
Please see last page of this advertisement for brief summary of prescribing information.

# ZOVIRAX® (acyclovir) CAPSULES

## Help free your patients from recurrences.

### Daily therapy

Coping with genital herpes is rarely easy. For some, the worst part is the pain and discomfort of frequent attacks —month after month, year after year. For others, the emotional burden presents a more difficult problem, leading to social isolation, anxiety, and diminished self-esteem.

### Prevent or reduce recurrences

Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

### Generally well tolerated

Daily therapy with ZOVIRAX CAPSULES is generally well tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/vomiting, vertigo, and arthralgia.

The physical and emotional difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episodes, as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRAX CAPSULES.

*Please see brief summary of prescribing information on next page.*

# Prevent recurrences month after month*

# ZOVIRAX® (acyclovir) CAPSULES

Brief Summary

**INDICATIONS AND USAGE:** Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psychosocial difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

**First Episodes** (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

**Recurrent Episodes:**

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

**CONTRAINDICATIONS:** Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

**WARNINGS:** Zovirax Capsules are intended for oral ingestion only.

**PRECAUTIONS: General:** Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

**Drug Interactions:** Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the $F_1$ generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

**Pregnancy:** ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

**Nursing Mothers:** It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

**Pediatric Use:** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS — Short-Term Administration:** The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

**Long-Term Administration:** The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

**DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes:** One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

**Chronic suppressive therapy for recurrent disease:** One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

**Intermittent Therapy:** One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

**Patients With Acute or Chronic Renal Impairment:** One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance $\leq 10$ ml/min/1.73/m$^2$.

**HOW SUPPLIED:** Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200"- Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

*In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

Wellcome

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Research Triangle Park
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# From RAP to RAPE and Beyond

A prophetic wag pointed out to me recently that the new rules proposing to make DRG lackeys of hospital-based radiologists, anesthesiologists, and pathologists might soon be extended to include emergency care physicians, adding that the acronym would then gain the status of an appropriately descriptive verb.

I can remember when I used to mourn the fact that we physicians were becoming civil servants. Although not many years have passed since my mourning period, I now realize there are things happening to us far worse than becoming civil servants. After all, civil servants have a union, can go on strike, have an impressive package of fringe benefits, have no liabilities, don't need liability insurance, can virtually never be fired, and answer to only one boss.

In comparison to being a RAP flunky in a hospital, holding my hat in my hand asking the administrator if the business manager has figured my share of the DRG payments yet, being a civil servant sounds dandy.

It won't be long, of course, until the acronym will be shortened to AP, signifying All Physicians who care for patients in hospitals. We'll become sharecroppers under the overseeing hospital administrators, forced to accept on faith their calculations of our earnings, or hire an auditor to challenge them.

Naturally, the DRG reimbursement schedule will not include a token for the hours we spend making the hospital work for the administrator; making it meet the standards and requirements for every group from the JCAH to the local fire department; staffing the dozens of committees; supervising and teaching medical students, residents, and a variety of other aspiring professionals; participating in the credentialing and privileging activities of the staff; and countless other volunteered activities.

No, there's not the slightest chance we'll be paid for any of this. We rarely receive a personal thank you from anyone. Our own bylaws, most of which are extracted from "suggestions" made by the hospitals' owners and the JCAH, tell us we have to agree to "volunteer" for these jobs in order to be a member of the staff.

Now, we are being told that the diagnoses we make will determine what the hospital will be paid for *our* services to *our* patients; this in addition to being told previously that only patients with certain diagnoses can be admitted to the hospital at all. And, if we make a mistake in guessing at an obscure illness, we could be "sanctioned" or fined by Medicare, and the hospital could not be paid for any of its services.

I wonder if, in such a case, the administrator would deduct the patient's bill from our share of the DRG income?

Yeah, I'd a lot rather be a civil servant than a sharecropper. But I wouldn't go to medical school to become either one.

*—MRJ*

Dear Fellow Physicians:

As I write my last letter to you as your president, I do so with mixed emotions.

First of all, I will be able to put more of my time into my practice and my patients which, after all, is my main interest. However, I have had tremendous pride and self-satisfaction engendered this past year by the trust of you, my peers, in me. The interesting people I have met and the many challenges of our profession that I have encountered have all been stimulating and a source of pleasure and satisfaction — sometimes frustrations. I only hope that I have been able to direct our society in a wise course for the future.

I feel I must share certain impressions with you. I am definitely optimistic where medicine is concerned. The vicissitudes and stresses that our profession is undergoing at this time are tending to create a natural selection of doctors, that is, those people who really want to be physicians in the true sense of the word.

Also, economically, we probably have even more cuts to face in our income and standard of living. But the attitudes of government and industry, I feel, are changing. These areas are no more friends than ever — and believe me, government, industry, business, and even hospitals are only friendly to us as long as our goals coincide with their needs — but these institutions are slowly beginning to realize the need to reward those people who dedicate their lives to the extensive training and work habits required to carry out the practice of medicine. They truly need our profession, and the message is beginning to sink in.

I do have two specific complaints and criticisms. Many among you are selfishly pursuing your own way and expecting others to fight your battles for you. Our involvement in this state is definitely increasing, as shown by an excellent turnout for the recent Medicine Day at the capitol, sponsored by our auxiliary, but too many physicians are still not involving themselves in the legislative and political process. If there is one thing I have learned this past year, it is that in this arena any hope for the future must begin.

I am also much disenchanted by the obvious self-aggrandizement, fee gouging, and likely even disinterest shown by some of our members. Granted, there are only a very few; however, it is these few doctors who create such a bad impression for all of us, for it hurts our efforts to obtain legislative, public, and political relief. Hopefully these members of our profession will realize the shortsightedness of their ways and join the majority in support of our entire effort.

We must continue to provide conscientious, skillful, and caring attention, but we must also accept some social fiscal responsibility in the future.

I do leave this office with a heavy heart. There are so many things to be done and so many problems to face. Our president-elect, Joe Crosthwait, I know will prove to be ever so capable; I only wish I could have done more to make his term even easier.

I end this not with goodbye, but with "hasta la vista." May God guide all of our efforts in a proper and wise direction.

Norman L. Dunitz, MD

# Barber-Surgeon Farmers

WARREN L. FELTON II, MD

Everyone has heard of barber-surgeons (eighteenth century) and gentlemen (surgeon) farmers (twentieth century). Both did well, while doing good, but the latter seem to have done better.

This discourse has to do with a somewhat different matter, however. Our American farmers are in trouble. Many have borrowed on inflated land prices and counted on inflated crop incomes to cover the debts. Both have dropped in value, much to the concern and detriment of the farmers and of the bankers who had the foresight to lend them money. This has a familiar ring to oilmen, restaurant and hotel investors, and other large and small businessmen.

But farmers are a special group. There is something sacred about the "family farm," to the extent that the federal government will subsidize farmers and actually pay them for not producing certain crops, or at least guarantee them a price for what they do choose to continue to produce.

This is an idea whose time has come. With the relative excess of surgeons and their probable drop in income, it seems clear that the federal government should subsidize surgeons. What real American would want the "family surgeon" to become extinct?

We've been led to believe that many of the surgical procedures now performed are unnecessary. Let's assume that this may be correct. The implantation of cardiac pacemakers has been cited by certain bureaucrats as a prime example of a procedure where 50% may be unnecessary. If this were true, how would we decide which patients are bona fide candidates?

I would suggest, to be fair, that every other patient referred for pacemaker implantation be denied a pacemaker. The surgeon, of course, would be paid (subsidized) at the usual fee for the procedure. But the savings in the cost of the pacemaker, operating room, and hospital charges would amount to a great deal, and still provide support for the "family surgeon."

What would become of the 50% of patients who did not get pacemaker implantation? They could be put in the now excessive hospital beds (we could call them silos) for storage. Those who didn't spoil in storage might still receive pacemakers when the price of the device (made in South Korea or Taiwan) became more affordable. The possible impact on the foreign trade deficit is another matter.

In the best of all possible arrangements, a gentleman-surgeon-farmer who acquired good wheat land could be paid both for not growing wheat and for not doing surgery.

The problem might be reduced a bit, as the farm problem has been, by a "family surgeon" concert or two by Willie Nelson. Whether it would actually save the "family surgeon" is not guaranteed. I would certainly attend, but then I'd probably go to a Willie Nelson concert even if it were given for the benefit of the Trial Lawyers' Association.

This is a matter which seems to require rather urgent action. If all of the "family surgeons" become gentlemen (subsidized) farmers, who would do the unnecessary, not to mention necessary, surgery?

As Willie would say, in song, "If you've got the money, Honey, I've got the time."

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# Cefotetan, an In Vitro Comparison with Other Antibiotics

S.M. HUSSAIN QADRI, PHD; KATHLEEN A. BELOBRAYDIC

**In vitro activity of cefotetan was tested against 2519 consecutive clinical isolates from a major teaching hospital in Oklahoma City. Gram negative facultative bacteria were comprised of 2055 strains; 355 strains of facultative gram positive and 109 isolates of anaerobic bacteria were used in this study. Antibacterial activity of cefotetan was compared with cephalothin, cefoxitin, and other commonly used antibiotics. Cefotetan demonstrated excellent activity towards *Escherichia coli*, Klebsiella, Enterobacter, Proteus, Providencia, Serratia, Citrobacter, and a variety of other facultative gram negative organisms. However, it had virtually no activity towards *Pseudomonas aeruginosa*. Of the 172 isolates of *Staphylococcus aureus* tested, 171 (99%) were susceptible to cefotetan. Seventy-one percent of all *S epidermis* tested were also sensitive to this new cephalosporin. It had excellent antibacterial activity against all streptococci except *Streptococcus fecalis*. Eighty-three percent of the 106 anaerobic bacterial isolates were also sensitive to cefotetan.**

Cefotetan is a new semisynthethic cephamycin which demonstrates a high degree of stability to hydrolysis by bacterial beta-lactamases.[1-3] It is a broad-spectrum antibiotic with activity against several common pathogenic aerobic and anaerobic bacteria. In vitro studies have shown that cefotetan is distinctly superior to first and second generation cephalosporins and comparable to other third generation cephalosporins.[4] In this paper we report the antibacterial activity of cefotetan towards 2500 clinical isolates of aerobic and anaerobic bacteria at Oklahoma Memorial Hospital, a major teaching hospital for the University of Oklahoma Health Sciences Center, Oklahoma City. Activity of cefotetan was compared with cephalosporin, cefoxitin, and other routinely used antibiotics.

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## Materials and Methods

**Microorganisms.** A total of 2519 consecutive isolates from clinical specimens at Oklahoma Memorial Hospital, Oklahoma City, were used in this investigation. They consisted of 2055 strains of aerobic or facultative gram negative rods, 355 facultative gram positive organisms, and 109 anaerobic bacteria. These isolates were obtained from blood cultures (7%), urines (32%), respiratory tracts (15%), and wounds, tissues, body fluids, and other miscellaneous sources (46%).

**Antimicrobial agents.** Commercially prepared antibiotic disks (Difco Laboratories, Detroit) were used during this investigation. Potency of antibiotics used were: 30 ug for cefotetan, amikacin, cephalothin, cefoxitin, chloramphenicol, nitrofurantoin, tetracycline, and vancomycin; 10 ug for ampicillin, gentamicin, and tobramycin; 15 ug for erythromycin; 100 ug for piperacillin and carbenicillin; 2 ug for clindamycin; 1.25/23.7 ug for trimethoprim-sulfamethoxazole; and 10 units for penicillin G. The disk content and interpretive data for cefotetan were provided by Stuart Pharmaceuticals, Wilmington, Del; for other antimicrobial agents, the

criteria established by the National Committee for Clinical Laboratory Standards[6] were used.

All the aerobic and facultative bacteria were tested by the standard conventional disk diffusion test.[6] Mueller-Hinton agar (Difco Laboratories), with a pH between 7.2 and 7.4, was used to perform disk diffusion susceptibility testing. Innoculated plates were incubated at 35°C for 18 to 24 hours. *E coli* ATCC 25922, *S aureus* ATCC 25923, and *P aeruginosa* ATCC 27853 were used as quality control organisms.

Antibacterial activity of cefotetan against anaerobic bacteria is shown in Table 3. All the isolates of clostridia and fusobacteria were inhibited by cefotetan. Of the 19 strains of penicillin-resistant *B fragilis*, 13 (66%) were susceptible to cefotetan. It was moderately active against other species of Bacteroides, propionibacteria, peptococci, and peptostreptococci.

## Discussion

Cefotetan is a new broad-spectrum cephamycin which resembles cephalosporins and is characterized by a methoxy group on 7-alpha-position. Its unique structure confers on it an increased stability to hydrolysis by many bacterial beta-lactamases.[3,7,8] In vitro activity of cefotetan has been extensively studied.[9]

Against *E coli*, the organism most commonly isolated from clinical specimens, cefotetan has been

**Table 1. Antibacterial Activity of Cefotetan and Other Antibiotics Against Gram Negative Aerobic or Facultative Bacteria**

| | Percent Susceptible | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Organisms (No. tested) | AN | AM | CB | CR | CFOX | C | GM | PIP | SXT | TM | CTT |
| *Escherichia coli* (364) | 100 | 67 | 70 | 86 | 98 | 98 | 99 | 90 | 93 | — | 101 |
| *Klebsiella pneumoniae* (146) | 100 | 15 | 7 | 94 | 97 | 96 | 99 | 98 | 92 | — | 100 |
| *K oxytoca* (7) | 100 | 10 | 10 | 65 | 100 | 100 | 100 | 38 | 100 | — | 100 |
| *K ozaenae* (4) | 100 | 25 | 25 | 75 | 100 | 100 | 100 | 100 | 100 | — | 75 |
| *Enterobacter cloacae* (62) | 100 | 46 | 92 | 16 | 15 | 98 | 100 | 96 | 97 | — | 88 |
| *E aerogenes* (29) | 100 | 21 | 76 | 28 | 4 | 95 | 100 | 100 | 96 | — | 83 |
| *E agglomerans* (10) | 92 | 46 | 67 | 25 | 25 | 83 | 100 | 100 | 83 | — | 100 |
| *Hafnia alvei* (15) | 100 | 10 | 45 | 0 | 13 | 33 | 95 | 95 | 65 | — | 40 |
| *Proteus mirabilis* (114) | 100 | 88 | 94 | 90 | 94 | 95 | 93 | 99 | 88 | — | 97 |
| *P vulgaris* (15) | 94 | 0 | 86 | 0 | 100 | 100 | 94 | 100 | 100 | — | 94 |
| *Providencia rettgeri* (4) | 100 | 25 | 100 | 25 | 75 | 75 | 100 | 100 | 100 | — | 100 |
| *P stuartii* (4) | 100 | 75 | 75 | 0 | 100 | 50 | 100 | 100 | 50 | — | 100 |
| *Acinetobacter calcoaceticus* var *anitratus* (11) | 100 | 6 | — | 0 | 0 | 11 | 100 | 100 | 22 | — | 0 |
| *A calcoaceticus* var *lwoffi* (4) | 100 | 100 | 100 | 0 | 0 | 100 | 100 | 100 | 0 | — | 100 |
| *Citrobacter diversus* (16) | 100 | 13 | 13 | — | 100 | 100 | 100 | 93 | 100 | — | 100 |
| *C freundii* (16) | 10 | 29 | 86 | 5 | 6 | 100 | 91 | 91 | 72 | — | 76 |
| *Morganella morganii* (18) | 100 | 8 | 93 | 3 | — | — | 96 | 100 | 93 | — | 100 |
| *Serratia marcescens* (39) | 98 | 15 | 100 | 4 | 52 | 90 | 100 | 96 | 100 | — | 98 |
| *S liquefaciens* (4) | 100 | 0 | 100 | 0 | 75 | 100 | 100 | 75 | 100 | — | 100 |
| *Psuedomonas aeruginosa* (121) | 80 | 4 | 66 | 0 | 2 | 0 | 85 | 85 | 0 | 95 | 4 |
| *P maltophilia* (4) | 50 | 50 | 100 | 0 | 0 | 100 | 50 | 100 | 100 | 0 | 100 |
| *Aeromonas hydrophilia* (4) | 100 | 0 | 0 | 50 | 50 | 100 | 100 | 100 | 100 | — | 100 |
| Alcaligenes sp (2) | 100 | 100 | 100 | 100 | 100 | 50 | 100 | 100 | 50 | — | 100 |
| Achromobacter sp (2) | 100 | 100 | 100 | 100 | 50 | 100 | 100 | 100 | 100 | — | 50 |
| Moraxella sp (4) | 100 | 75 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | — | 100 |
| Flavobacterium sp (2) | 50 | 0 | 50 | 0 | 100 | 100 | 50 | 100 | 0 | — | 50 |
| *Pasteurella multocida* (2) | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | — | — | 100 |
| *Eikenella corrodens* (2) | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | — | — | 100 |

**AN** = amikacin, **AM** = ampicillin, **CB** = carbenicillin, **CR** = cephalothin, **CFOX** = cefoxitin, **C** = chloramphenicol, **GM** = gentamicin, **PIP** = piperacillin, **SXT** = trimethoprim-sulfamethoxazole, **TM** = tobramycin, **CTT** = cefotetan

Table 2. Antibacterial Activity of Cefotetan Against Gram Positive Bacteria

| Organisms (No. tested) | Percent Susceptible | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | AM | CR | CC | C | E | GM | ME | FD | P | TE | Va | CTT |
| *Staphylococcus aureus* (172) | 5 | 98 | 96 | 98 | 96 | 95 | 98 | 100 | 5 | 87 | 100 | 99 |
| *S epidermidis* (83) | 28 | 98 | 73 | 85 | 55 | 76 | 98 | 98 | 28 | 64 | 100 | 71 |
| *S saprophyticus* (6) | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| *Streptococcus pyogenes* (12) | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| *S agalactiae* (16) | 100 | 100 | 100 | 100 | 100 | 50 | 100 | 100 | 100 | 5 | 100 | 100 |
| *S pneumoniae* (21) | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| *S fecalis* (19) | 88 | — | — | — | 100 | — | — | — | 11 | 11 | 100 | 11 |
| Viridans streptococci (15) | 100 | 100 | 100 | 100 | 93 | 100 | 86 | 100 | 100 | 52 | 100 | 86 |
| Nonhemolytic strep (4) | 75 | 50 | 50 | 100 | 75 | 50 | 50 | 75 | 50 | 75 | 75 | 100 |
| Corynebacterium sp (7) | 88 | 100 | 71 | 100 | 100 | 88 | 88 | — | 88 | 88 | 100 | 100 |

**AM** = ampicillin, **CR** = cephalothin, **CC** = clindamycin, **C** = chloramphenicol, **E** = erythromycin, **GM** = gentamicin, **ME** = methicillin, **FD** = nitrofurantoin, **P** = penicillin, **TE** = tetracycline, **Va** = vancomycin, **CTT** = cefotetan

reported to have activity comparable to moxalactam, ceftazidime, and cefotaxime, and superior to cefoxitin and cefoperazone.[7,8,10,11] Similar findings have been reported for klebsiella species and other members of Enterobacteriaceae.[12-15] At Oklahoma Memorial Hospital we found that 520 of the 521 isolates of *E coli* and klebsiella species were susceptible to cefotetan. This activity was greater than those of eight other antibiotics commonly used in the USA.

Overall inhibitory effect of cefotetan against gram negative bacteria was superior to other antibiotics, with all the organisms tested except *P aeruginosa, Acinetobacter calcoacetius* var *anitratus*. Only 4% of the 121 strains of *P aeruginosa* and none of the 11 strains of *A calcoacetius* var *anitratus* tested were inhibited by cefotetan. Other investigators have reported similar observations, noting that unlike some other third generation cephalosporins, cefotetan has little activity against these organisms.[7,10-13,16]

*S aureus, S saprophyticus, S pyogenes, S agalactiae, S pneumoniae,* and viridans streptococci were extremely sensitive to cefotetan. Chattopadhyay and Teli[17] and Russo et al[11] reported that some strains of *S fecalis* are moderately sensitive to cefotetan. However, we found that only 11% of our isolates of this organism were inhibited by cefotetan, an observation that is consistent with those reported by Ward and Richards[8] and Hennessey.[9] The antibacterial spectrum of cefotetan appears to be similar to that of moxalactam towards gram positive bacteria.[8,18]

Cefotetan exhibited excellent activity against clostridia and fusobacteria, and moderate activity against *B fragilis* and anaerobic cocci. Hennessey[9] reported that almost 94% of *B fragilis* strains were inhibited by cefotetan; we found that only 66% of

Table 3. Activity of Cefotetan Against Anaerobic Bacteria

| Organisms (No. tested) | Percent Susceptible | | | | | |
|---|---|---|---|---|---|---|
| | CR | C | E | P | TE | CTT |
| Peptostreptococci (14) | 94 | 100 | 89 | 94 | 73 | 73 |
| Peptococci (20) | 92 | 96 | 85 | 79 | 77 | 83 |
| Propionibacteria (26) | 92 | 97 | 92 | 92 | 86 | 85 |
| *Bacteroides fragilis* (19) | 100 | 100 | 100 | 0 | 66 | 66 |
| *B melaninogenicus* (4) | 100 | 50 | 100 | 25 | 100 | 100 |
| *B oralis* (2) | 100 | 100 | 100 | 100 | 100 | 100 |
| *Clostridium perferingens* (11) | 100 | 100 | 100 | 100 | 100 | 100 |
| *C ramosum* (4) | 100 | 100 | 0 | 0 | 100 | 100 |
| Fusobacterium sp (9) | 100 | 100 | 100 | 100 | 100 | 100 |

**CR** = cephalothin, **C** = chloramphenicol, **E** = erythromycin, **P** = penicillin, **TE** = tetracycline, **CTT** = cefotetan

strains at Oklahoma Memorial Hospital were susceptible to this antimicrobial agent.

Members of Enterobacteriaceae, *S aureus*, clostridia, Bacteroides species, and anaerobic cocci constitute a vast majority of organisms isolated from clinical specimens of infected patients. Polymicrobial etiology is suspected in 6% to 8% of infections, involving both aerobic and anaerobic bacteria. Since many of these organisms were found to be susceptible to cefotetan, it offers a useful addition to the valuable group of antibacterial drugs used in the treatment of such infections.

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## Coming in May . . .

Manuscripts in production for the May OSMA JOURNAL include an overview of infantile autism, a study of the occurrence of bacteria at a VA medical center, and a psychiatric report on a child-abusing parent. Reaction Time will feature a letter from Dr Joe D. Haines, Jr., responding to last month's criticism of his December article, "The Ubiquitous Pharmaceutical Representative."

# Augmentation Enterocystoplasty in Children with Myelomeningocele

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**We report our experience with twelve children with myelomeningocele and neurogenic bladder who underwent bladder augmentation. Of the seven children who underwent the procedure to improve continence, six (86%) have satisfactory levels of continence on intermittent clean catheterization. The remaining five children had bladder augmentation in conjunction with primary undiversion. Three (60%) of these patients have had satisfactory results. We believe that bladder augmentation, when combined with a program of intermittent clean catheterization, is an effective procedure to restore continence in children with myelomeningocele and poorly compliant bladders.**

Children with myelomeningocele often develop low-volume and low-compliance contracted bladders, which results in incontinence, recurrent urinary tract infections, or upper tract dysfunction. Previously, urinary diversion was a mainstay in the treatment of children with these problems, but with the advent of clean intermittent catheterization and newer pharmacologic agents, many children no longer require surgery. There remains a subset of patients in whom intermittent catheterization and pharmacologic manipulation is inadequate to prevent the complications of a neurogenic bladder. Augmentation enterocystoplasty has been used successfully to increase bladder capacity and to increase compliance in patients with contracted bladders resulting from interstitial cystitis, tuberculous cystitis, prior surgery, trauma, or neurologic disease.

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## Materials and Methods

We reviewed the records of twelve children who have undergone bladder augmentation at this institution since 1980. Five children were male and seven female; ages at the time of surgery ranged from six to sixteen years. Eight children had thoracolumbar or lumbar level myelomeningocele lesions and four had either sacral lesions or sacral agenesis.

Indications for operation are shown in Table 1. Bladder augmentation was the sole procedure in four children. Augmentation was combined with other procedures in eight children and these are shown in Table 2. Bladder reconstruction was accomplished by one of four procedures and these included:

**Table 1. — Indications for Operation**

| | |
|---|---|
| Undiversion | 5 |
| Improve continence | 6 |
| Correction of reflux | 2 |
| Recurrent UTI | 2 |
| Deterioration of renal function | 1 |

ileocecocystoplasty (4), cecocystoplasty (3), ileocystoplasty or ileal cup patch cystoplasty (4), or sigmoid patch cystoplasty (1).

Prior to surgery, complete histories were obtained from all patients and all underwent physical examination, routine laboratory work, either an excretory urogram or nuclear renal scan, and a urodynamic evaluation. Postoperatively, all patients have been followed with BUN and creatinine levels and either an excretory urogram or renal scan. Ten patients have had postoperative urodynamic evaluations and either voiding cystourethrograms or nuclear cystograms. All but one child (or parents) were interviewed postoperatively.

Length of follow-up ranges from six months to five and one-half years. One child was followed for three years postoperatively but was then lost to follow-up and was not interviewed.

## Results

Of the six children who underwent augmentation to improve continence, five have had satisfactory results. Four are completely dry on intermittent clean catheterization and do not require additional pharmacologic bladder manipulation. One child continues to wear pads and to require oxybutinin. Among these children, bladder volume, as measured by urodynamics, has increased from 55% to over 1000% over baseline, and compliance has markedly improved. One child, whose indication was to improve continence, has had repeated postoperative urinary tract infections despite frequent catheterization and suppressive antibiotics. He and his family moved shortly after surgery. His postoperative excretory urogram was normal, as was his postvoid residual. He has had no urodynamic evaluations since surgery, and he is improving on antibiotics and oxybutinin.

One child in this group suffered a perioperative leak from his transureteroureterostomy site and required further surgery for repair. Subsequently, he has done well without further complication.

One female child underwent bladder augmentation because of recurrent infections and decreased renal function. Preoperatively, she was felt to have functional ureterovesical obstruction secondary to a poorly compliant, hypertrophied bladder wall. After augmentation her bladder compliance improved and bladder volume increased by 35%. She has remained infection free, and renal function has improved.

Five children underwent bladder augmentation as part of an undiversion procedure. Three children have had satisfactory results, with stable renal functions and adequate bladder capacities, and are completely dry on intermittent catheterization. Two patients are over twenty years old and catheterize themselves only two to three times per day, without complicating infections or leakage of urine.

Table 2. — Procedures Done in Addition to Augmentation Enterocystoplasty

| | |
|---|---|
| Vesicopexy/urethropexy | 3 |
| Transureteroureterostomy | 2 |
| Ureteroneocystostomy | 1 |
| Resection of diverticulum | 1 |
| Takedown of reservoir/conduit | 4 |
| Implantation of artificial urinary sphincter | 2 |
| Takedown cutaneous ureterostomies | 1 |

Two children had poor results. The first child had a takedown of an ileocecal reservoir with cecocystoplasty and placement of an artificial urinary sphincter. Postoperatively, the child, a male, failed to perform catheterization with adequate frequency and subsequently moved to the care of another physician. He has had recurrent urinary tract infections, removel of his sphincter due to infection, and subsequent placement of a suprapubic catheter. The second child underwent a takedown of an ileal conduit, ileocecocystoplasty, and placement of an artificial urinary sphincter. Postoperatively, the patient experienced progressive upper urinary tract deterioration and underwent rediversion surgery. His renal function stabilized, and he eventually underwent undiversion surgery again.

Thus far there have been no complications attributable to the enterocystoplasty procedure itself.

## Discussion

Over the past several years, bladder augmentation enterocystoplasty has become increasingly employed in the surgical correction of the contracted, neurogenic bladder.[1-4] Oklahoma Children's Memorial Hospital has an increasingly large population of children with myelomeningocele who are closely followed at periodic intervals for evidence of changes in renal function and for assessment of their voiding dysfunction. We continue to find children in this group who fail trials of clean, intermittent catheterization and pharmacologic therapy for the complications of neurogenic bladder, and who thereby require bladder augmentation.

We have used bladder augmentation in patients with small contracted and poorly compliant bladders,

in an effort to preserve renal function, to improve or restore continence, and in conjunction with urinary undiversion. Although our first five cases of augmentation were in patients requiring undiversion, our experience, like others,[5] has persuaded us to shift towards early reconstruction instead of early diversion. Our success rate of 60% is similar to others who have submitted children with myelodysplasia to undiversion surgery.[6] Likewise, our success rate of 80% in the group undergoing augmentation as a primary procedure is similar to others;[7] we have also noted the higher success rate in the early reconstruction group, as opposed to the undiversion group.[8]

The importance of preoperative patient preparation and selection is vital; at least one of our failures was attributable to the child's unwillingness to catheterize himself. The importance of this issue has previously been noted by Kass[9] and Bauer.[10] The myelomeningocele children in our group are generally well suited to their own roles in postoperative care, however, because many are on a program of clean, intermittent catheterization. Continence in these children also depends on sphincter function, and its preoperative assessment is equally important.[10] Augmentation enterocystoplasty can easily be combined with artificial sphincter placement to restore continence.[3]

## Summary

We find augmentation enterocystoplasty an effective procedure in children with neurogenic, contracted bladders that are unresponsive to other treatment. Success rates are better in children not previously subjected to diversion surgery, which gives this procedure an important role in the early management of children with this handicap. This procedure, however, is also effective in those children previously submitted to diversion surgery who wish to have urinary tract continuity restored. A postoperative catheterization program that is strictly followed is mandatory if long-term success is to be achieved.

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*Vincent Michael Fiorica, MD, was graduated from the University of Oklahoma College of Medicine in 1983. He was a fourth year resident in urology when this paper was written.*

*William F. Barnes, MD, is a clinical assistant professor in the urology department at the OU College of Medicine and has a private practice in Oklahoma City. The 1969 graduate of the University of Texas Medical Branch at Galveston is a member of the American Urological Association, Society for Pediatric Urology, and Urodynamic Society.*



**May 1 is the closing date for the June 1987 OSMA JOURNAL**

News from
the Oklahoma State
Department of Health

## Seat Belt Project

Automobile accidents are the leading cause of death for teenagers and for adults under age thirty-five. These tragic deaths could be greatly reduced if Oklahomans learned to consistently do one simple healthy thing — buckle a seat belt. Because of its emphasis on prevention, the Oklahoma State Department of Health (OSDH) was awarded funding for seat belt education through the Oklahoma Highway Safety Office. The primary populations targeted to receive seat belt education include high school students and clients of local county health departments.

The high school program was developed so the major responsibility of implementing the program rests upon the students. An OSDH health educator presents the program to a student group, such as the student council, and provides instruction on how to start the program. Then the OSDH health educator coordinates and participates in the main seat belt education assembly for the students. All other aspects of the program, including data collection, promotion, news releases, and incentive collection and distribution, are the reponsibility of the students, with technical guidance from the health educator. During the first year of the contract, seven such high school projects were implemented, reaching 5,384 students.

The county health department program also uses a number of approaches designed to influence clients using county health department clinics. A special effort is made to provide education to those who are pregnant or have small children. In addition to the distribution of pamphlets and other materials, seat belt education is provided through local county health department staff presentations and counseling, as well as audiovisual presentations in the department waiting areas. Also, for women with infants or toddlers, loaner car safety seats are made available with instruction on their proper use.

Physicians who would like more information about the seat belt education programs may contact Neil E. Hahn, MPH, Health Education and Information Service, 405-271-5601.

| DISEASE | January 1987 | TOTAL TO DATE | | |
|---|---|---|---|---|
| | | This Year | Last Year | 5 Yr. Avg. |
| AMEBIASIS | 2 | 2 | 1 | 0 |
| CAMPYLOBACTER INFECTIONS | 16 | 16 | 12 | — |
| ENCEPHALITIS, INFECTIOUS | 1 | 1 | 0 | 1 |
| GIARDIA INFECTIONS | 14 | 14 | 15 | — |
| GONORRHEA (Use ODH Form 228) | 865 | 865 | 1186 | 1118 |
| HAEMOPHILUS INFLUENZAE INVASIVE DISEASE | 6 | 16 | 19 | — |
| HEPATITIS A | 24 | 24 | 19 | 22 |
| HEPATITIS B | 19 | 19 | 5 | 8 |
| HEPATITIS, NON-A NON-B | 4 | 4 | 1 | — |
| HEPATITIS UNSPECIFIED | 7 | 7 | 5 | 5 |
| MEASLES (RUBEOLA) | 0 | 0 | 0 | 0 |
| MENINGITIS, ASEPTIC | 4 | 4 | 4 | 3 |
| MENINGITIS, BACTERIAL (non-meningococcal, non H. Influenzae) | 4 | 4 | 1 | 2 |
| MENINGOCOCCAL INFECTIONS | 2 | 2 | 3 | 1 |
| PERTUSSIS | 2 | 2 | 1 | 1 |
| RABIES (Animal) | 2 | 2 | 4 | 7 |
| ROCKY MOUNTAIN SPOTTED FEVER | 3 | 3 | 0 | 0 |
| RUBELLA | 0 | 0 | 0 | 0 |
| SALMONELLA INFECTIONS | 16 | 16 | 21 | 14 |
| SHIGELLA INFECTIONS | 23 | 23 | 10 | 11 |
| SYPHILIS (Use ODH Form 228) | 14 | 14 | 18 | 14 |
| TETANUS | 0 | 0 | 0 | 0 |
| TUBERCULOSIS | 9 | 9 | 6 | 14 |
| TULAREMIA | 3 | 3 | 0 | 0 |
| TYPHOID FEVER | 0 | 0 | 0 | 0 |

| Diseases of Low Frequency | Total to Date This Year |
|---|---|
| ACQUIRED IMMUNE DEFICIENCY SYNDROME | 0 |
| BRUCELLOSIS | 0 |
| LEGIONNAIRES DISEASE | 2 |
| MALARIA | 0 |
| REYE SYNDROME | 1 |
| TOXIC SHOCK SYNDROME | 0 |

# "I thought I was totalled..."

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**Medicine Day, February 18,** at the state capitol was considered a big success by its organizers, the Oklahoma State Medical Association and the OSMA Auxiliary. Several hundred physicians, physician spouses, and friends of medicine gathered at the capitol to meet with their legislators and to learn about the legislative process. As seen here, the registration desk and exhibits were on the fourth floor rotunda and luncheon was served on the fifth floor, above.

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*ABC Close Up "Diagnosis: Malpractice"*

# ABC guilty of sensationalizing malpractice crisis, says AMA VP

The American Medical Association (AMA) has taken a national television network to task for its lack of objectivity in presenting a Saturday, December 27, program that sensationalized the complex professional liability problem.

The program was an ABC Close Up presentation entitled "Diagnosis: Malpractice." Although the holiday season program had low viewership, many physicians have expressed concern to the AMA about the telecast's shallowness and lack of balance.

In a letter to Richard Richter, senior producer for ABC News Close Up, James S. Todd, MD, AMA's senior deputy executive vice president, pinpointed the show's major shortcomings and urged that Close Up develop a second program to place the professional liability crisis, its causes and its potential solutions, in proper perspective for the public.

The full text of Dr Todd's letter follows:

Dear Mr. Richter:

Whatever the intent may have been, your recent ABC Close Up presentation entitled *Diagnosis: Malpractice* succeeded only in sensationalizing a very complex problem. The viewer was left with an inaccurate and frightening impression that each encounter with a physician is fraught with great danger. A great disservice is done by creating an atmosphere of distrust and fear among patients. The brief comment early in the program that the malpractice crisis is not the result of poor quality care was lost in the visual impact and emotional reporting of isolated incidents.

Having contributed to your research data, we are at a loss to explain the absence of objectivity. We do not deny that negligence exists, but not to the degree indicated by the current level of litigation or the increasing level of awards made by juries.

Physician discipline was given scant credit despite the joint actions of the Federation of State

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## ABC sensationalizes (continued)

Medical Boards and the AMA to identify and eliminate errant physicians. Congressman Wyden was given great credit, ignoring the fact that organized medicine was instrumental in helping draft and pass his legislation.

Insurance companies, highly regulated, were treated shabbily, especially by Robert Hunter, who repeatedly confuses insurance issues and is a spokesperson for the trial bar. You and he both have neglected to study physician owned, nonprofit monoline insurance companies that clearly have suffered intolerable loss ratios.

Totally omitted were the inequities of a judicial system where sixty percent of cases filed are without merit; fifty percent of dollars awarded go to five percent of the cases; and sixty percent of premium dollars are consumed by the system and never reach injured patients or physicians in the current system.

We would have preferred that your program give some mention to the federal and state efforts to bring about changes in the tort system that preserve patients rights while at the same time protecting hospitals and physicians against unwarranted litigation and excessive economic penalties. The cost of insurance under the current system is causing many physicians to curtail or leave their practices. One result is that some medical care, particularly in obstetrics, may be in short supply.

Medical care is an inexact science. Under the best of circumstances, unfortunate results occur. Our current system fails adequately to distinguish between unfulfilled expectations and negligence. Your program missed the opportunity to educate health care consumers on what they can do to protect themselves from substandard care, and how the current level of litigation is diminishing the availability of needed care, inflating the cost of health care and stifling innovative technology.

Given the huge number of patient-physician encounters every day, the medical profession can be proud of its accomplishments. At the same time the medical care system has some shortcomings that physicians and their professional organizations are working hard to correct. We would urge you to schedule a second broadcast to complete the liability picture, and we would be pleased to contribute to the content of a second program.

Sincerely yours,
*James S. Todd, MD*

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Top: Larry L. Long, MD, speaker of the OSMA House of Delegates, addresses the physicians assembled in the House chamber. Left, t to b: Rep John W. Bumpus, MD, (R-Bethany); House majority leader Guy Davis (D-Calera); and Senate President Pro Tem Rodger A. Randle (D-Tulsa) at the podium. Above: After making his opening remarks, OSMA President Norman L. Dunitz, MD, (left) greets Governor Henry Bellmon.

Medicine Day M.D.

*Medical Doctors Promoting Good Health*

# Medicine Day a capitol event

Doctors had their day — Medicine Day — at the state capitol in February, and what a day it was. Wearing their distinctive red badges, they and their spouses and friends came from all corners of the state February 18 to exchange ideas with each other and with their legislators.

Medicine Day, a joint project of the OSMA and the OSMA Auxiliary, had several purposes. These included familiarization with the legislative process and the issues being addressed in this session, promotion of the medical profession, and the development of lines of communication with legislators for future use.

Top and middle: Doctors, auxiliary members, and friends of medicine pack the gallery and floor of the House chamber for the morning program. Bottom: Joseph W. Stafford, MD, Enid, takes the floor.

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**Medicine Day registration** draws a crowd on the fourth floor rotunda at the state capitol, above. Already registered, William S. Harrison, MD, Chickasha, moves through the crowd with his morning coffee, being served nearby. Left, lunch is served on the fifth floor.


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## Board of Trustees approves eleven OSMA Life Memberships

Eleven Life Memberships were approved by the Oklahoma State Medical Association's Board of Trustees at their February 15 meeting.

The new Life Members are Richard W. Payne, MD, and Robert I. Trent, MD, Oklahoma City; Frank W. Clark, MD, and Ruben H. Mayberry, MD, Ardmore; Robert G. Allen, MD, and John E. Scott, MD, Bartlesville; John A. Graham, MD, Pauls Valley; Jake Jones, Jr., MD, Shawnee; Herbert B. Shields, Jr., MD, Enid; Milton J. Sugarman, MD, Elk City; and Denton B. Thomas, MD, Tulsa.

To be eligible for a Life Membership, an OSMA member must meet one or more of the following qualifications: (1) Be retired from the active practice of medicine due to age or ill health; (2) Be engaged in the active practice of medicine for fifty years or more; (3) Be seventy years of age or older.


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**William L. Hughes, MD,** (left) receives a plaque of appreciation for his ten years of service on the OSMA Council on State Legislation. The presentation is made by OSMA President Norman L. Dunitz, MD, at the February 15 meeting of the OSMA Board of Trustees.

*Wearing it inside and out*

## OKC surgeon uses GORE-TEX to replace damaged knee ligament

GORE-TEX, a synthetic fiber used in running and outdoor gear, has recently been used in Oklahoma for the first time in orthopedic surgery. The surgeon was David L. Holden, MD, of Oklahoma City.

He used the GORE-TEX material to replace the anterior cruciate ligament of a man in his twenties. Dr Holden has since performed another such surgery, and the postoperative prognosis for both patients remains excellent.

The use of GORE-TEX in this manner was approved by the Food and Drug Administration in October, 1986.

Why GORE-TEX? Dr Holden pointed to two of its basic properties, strength and porosity. "The GORE-TEX ligament is five times as strong as its biological counterpart," he said, "and its porous nature greatly enhances bone ingrowth. In nonmedical terms, this latter property means that the GORE-TEX ligament easily becomes part of the bone to which it has been affixed."

Dr Holden also emphasized the fiber's weight bearing and characteristic resilience and their role in markedly reducing rehabilitation time. He added that the GORE-TEX prosthesis would not be appropriate for older patients for whom a full knee replacement would more typically be indicated.

Dr Holden prefers to accomplish the implant arthroscopically.

*Media causing "unnecessary anxiety"?*

# AIDS in heterosexuals called a cause for concern, not panic

Heterosexual transmission of AIDS is a cause for concern but not a reason for panic, according to articles in the January/February issue of *ACSH News & Views*, a publication of the American Council on Science and Health (ACSH), an independent scientific organization.

"In their well meaning attempts to rivet attention on the potential future dangers of heterosexual transmission of AIDS, some public health authorities and the media covering their pronouncements may be causing unnecessary anxiety," said ACSH Executive Director Elizabeth M. Whelan, ScD.

"Sexually active Americans outside monogamous relationships do need to be made aware that AIDS can be transmitted heterosexually. The *potential* danger is real," she said. "But they should also be informed that both AIDS and evidence of infection with the AIDS virus are *currently* very rare in the non-drug-using heterosexual population. Heterosexuals may need to modify their future sexual behavior, but they need not panic over past relationships.

"Although caution is certainly warranted, at this point it is not in the interest of sound mental health and overall well being to terrify and demoralize people about past sexual encounters which appear to pose only remote risks," Dr Whelan said.

ACSH recommends that heterosexual men and women avoid having multiple and/or anonymous sex partners, including prostitutes. Persons choosing to have sex with someone who may be infected with the AIDS virus or whom they do not know well should use a condom at all times, preferably in combination with a spermicide, ACSH advises.

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*Age alone should not preclude giving blood*

## Studies support autologous blood donation and donors over age 65

Contrary to current practices, healthy persons aged 65 years and older should be encouraged to consider donating blood, according to a report in the *Journal of the American Medical Association*. The study found no increase in adverse reactions among older donors compared with younger ones. A second study in the same issue documents the benefits of autologous blood donation, the practice of donating one's own blood ahead of time for scheduled surgery.

"The notion that it is unsafe for older persons to donate blood is not confirmed by the clinical evidence," say Johanna Pindyck, MD, of the Greater New York Blood Program, and colleagues. They conducted a controlled study to measure the effect of blood donation on volunteers aged 66 to 78 years compared with those aged 52 to 65 years. "Eight of 504 older donors had a reaction, as compared with seven of 510 younger donors," the researchers say. All but one reaction in each group were defined as mild: pallor, dizziness, increased respiratory rate, or other temporary changes.

The growing elderly population uses a disproportionate amount of the nation's blood supply, the researchers observe. "At present there are approximately 30 million Americans older than 65, a large group that will grow to greater than 50 million by 2020," they say. "Although they represent approximately 12% of the population and a minute fraction of the donor pool . . . the elderly account for greater than 25% of blood use."

Donor age as a criteria for blood donation may depend on regional or state regulations, the study notes. The American Association of Blood Banks suggests that donors 66 years and older may be accepted at the discretion of the blood bank physician. Until recently the American Red Cross has suggested that donor sites require written consent from a personal physician of those older than 65.

Some elderly persons may be more susceptible to transient hypotension or other effects of blood donation, the researchers say. "However, counteracting these concerns is the positive selection factor that


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can be expected to occur: older donors will likely be a self-selected group who have donated before, have positive feelings about blood donation, and are motivated to continue in that capacity. . . . More important, the elderly who choose to continue donating after the age of 65 will be those who feel healthy enough to do so." Being denied the opportunity to give blood may be harmful psychologically, the report adds, since it represents a lost means of contributing to society.

In a second study, Roger K. Haugen, MD, and Gordon E. Hill, MD, describe a successful ten-year program for autologous blood donation at Holy Cross Hospital, Fort Lauderdale, Fla. To avoid the hazards of homologous blood transfusions during major orthopedic surgical procedures, 1,672 patients donated 6,615 units of blood. The majority of blood components were stored frozen, and later used in 1,938 surgical procedures, accounting for 95% of the transfusion requirements. Intraoperative blood salvage was also used, allowing 934 patients to be reinfused with their own blood at the time of surgery.

Interest in starting the program came from one of the authors who had had two deaths from post-transfusion hepatitis, following major hip surgery, according to the report. "The incidence of post-transfusion hepatitis in prospectively followed up, multiply transfused patients is approximately 10%," the researchers observe.

A patient's age alone does not preclude his being a blood donor, the study notes: 81.6% of the donors in the program were older than 60 years and 162 donors (8.4%) were between 80 and 91 years of age. "The large-scale use of autologous blood is considered feasible, practical, and cost efficient, has contributed to the community's blood supply, and provides the patient with the safest blood available — his own blood," the report concludes.


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**Tracy Bryan,** of television station KWTV, Oklahoma City, interviews OSMA President Norman L. Dunitz, MD, at the state capitol during Medicine Day.


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**Present and accounted for** at the state capitol on Medicine Day are (l to r) Donald L. Cooper, MD, Stillwater; James B. Eskridge III, MD, past OSMA president; Norman L. Dunitz, MD, OSMA president; and Kelsey Walters, OSMA Auxiliary president.

## In Memorium

### 1986

| | |
|---|---|
| *Alexander Poston, MD* | *January 3* |
| *Francis M. Duffy, MD* | *February 5* |
| *Edward L. Leonard, MD* | *February 14* |
| *William C. Tisdal, MD* | *February 24* |
| *Donovan Dillon Mosher, MD* | *April 4* |
| *Fred D. Switzer, MD* | *May 10* |
| *John D. Jennings, MD* | *May 12* |
| *Phillip Wade Jones, MD* | *May 18* |
| *Herbert L. Owen, MD* | *May 28* |
| *Marianne Elsbeth Kosbab, MD* | *June 13* |
| *William W. Rucks, Jr., MD* | *June 27* |
| *Ralph A. Smith, MD* | *July 27* |
| *Howard D. Tuttle, MD* | *August 3* |
| *Welborn W. Sanger, MD* | *September 19* |
| *William Carl Ewell, MD* | *September 20* |
| *Marcella Steel, MD* | *October 1* |
| *Terry Dwight Leming, MD* | *October 13* |
| *William Pat Fite, Jr., MD* | *October 30* |
| *Samuel Jackson McDaniel, MD* | *November 2* |
| *Iron Hawthorne Nelson, MD* | *November 12* |
| *John Robert Walter Spencer, MD* | *December 4* |

### 1987

| | |
|---|---|
| *Edward Leon Moore, MD* | *February 14* |
| *Ralph Cameron Emmott, MD* | *February 16* |
| *James Laurel Haddock, Jr., MD* | *February 19* |

## Deaths

### Ralph Cameron Emmott, MD
### 1921 - 1987

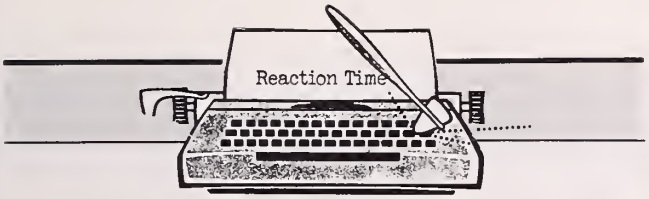
OSMA Life Member Ralph C. Emmott, MD, a urologist, died February 16, 1987. The Bartlesville physician was a native of Brantford, Ontario, Canada, and earned his medical degree at the University of Western Ontario, where he was graduated in 1945. Dr Emmott practiced medicine in Stilwell for ten years before moving to Bartlesville. He served with the US Air Force from 1979 to 1983.

### James Laurel Haddock, Jr., MD
### 1906 - 1987

Abdominal surgeon and gynecologist James L. Haddock, Jr., MD, of Norman died February 19, 1987. Dr Haddock was born in Whitecastle, La, raised in Oklahoma, and graduated from the University of Oklahoma School of Medicine in 1932. He established his medical practice in Norman in 1934 and retired in 1983.

### Edward Leon Moore, MD
### 1914 - 1987

Tulsa general surgeon Edward L. Moore, MD, a Life Member of the OSMA, died February 14, 1987. A 1939 graduate of St Louis University School of Medicine, he established his practice in Tulsa in 1947. During World War II, Dr Moore was on active duty with the US Army for 47 months and held the rank of major at the time of his discharge. The Uniontown, Pa, native was a Fellow of the American College of Surgeons.




## From Ada: Stop supporting OFPR

*To the Editor:* I am writing you for two reasons. I want to applaud your stand on the editorial page of the JOURNAL of the Oklahoma State Medical Association [Jan 87] against having Oklahoma physicians administer these insane regulations under which we are required to practice. I agree with you that we should quit "doing it to ourselves."

I was prompted to write at this particular time as I just received a denial for a patient recently discharged from the hospital here in Ada. It is becoming so obvious that these rules which we, as physicians, are administering unto ourselves are primarily and foremost to cut down on the cost of hospitalization; and the patient, physician, and hospital be damned.

It is refreshing to finally see a physician such as yourself with statewide prestige taking the stand you are taking. I vote to withdraw the support of the Oklahoma State Medical Association for the OFPR.

*Thomas R. Johnston, MD*
*Ada*

## From OKC: "Golden Years" a hit

*To the Editor:* I just wanted to write you a note to let you know how much I appreciate your editorials in the state medical journal. I always read them and enjoy them, but I think this last one ["Ah — The Golden Years," Jan 87] was exceptionally appropriate.

Please keep up the good work.

*H. C. Dodson, Jr., MD*
*Oklahoma City*

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**At the capitol** for Medicine Day: Jerry L. Puls, MD, Tulsa; James R. Priest, MD, Pawhuska; and Michael L. Wizenread, MD, Oklahoma City.

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Enclose payment with your ad and mail to: OSMA JOURNAL, 601 Northwest Expressway, Oklahoma City, OK 73118. OSMA members and state agencies will be invoiced upon request.

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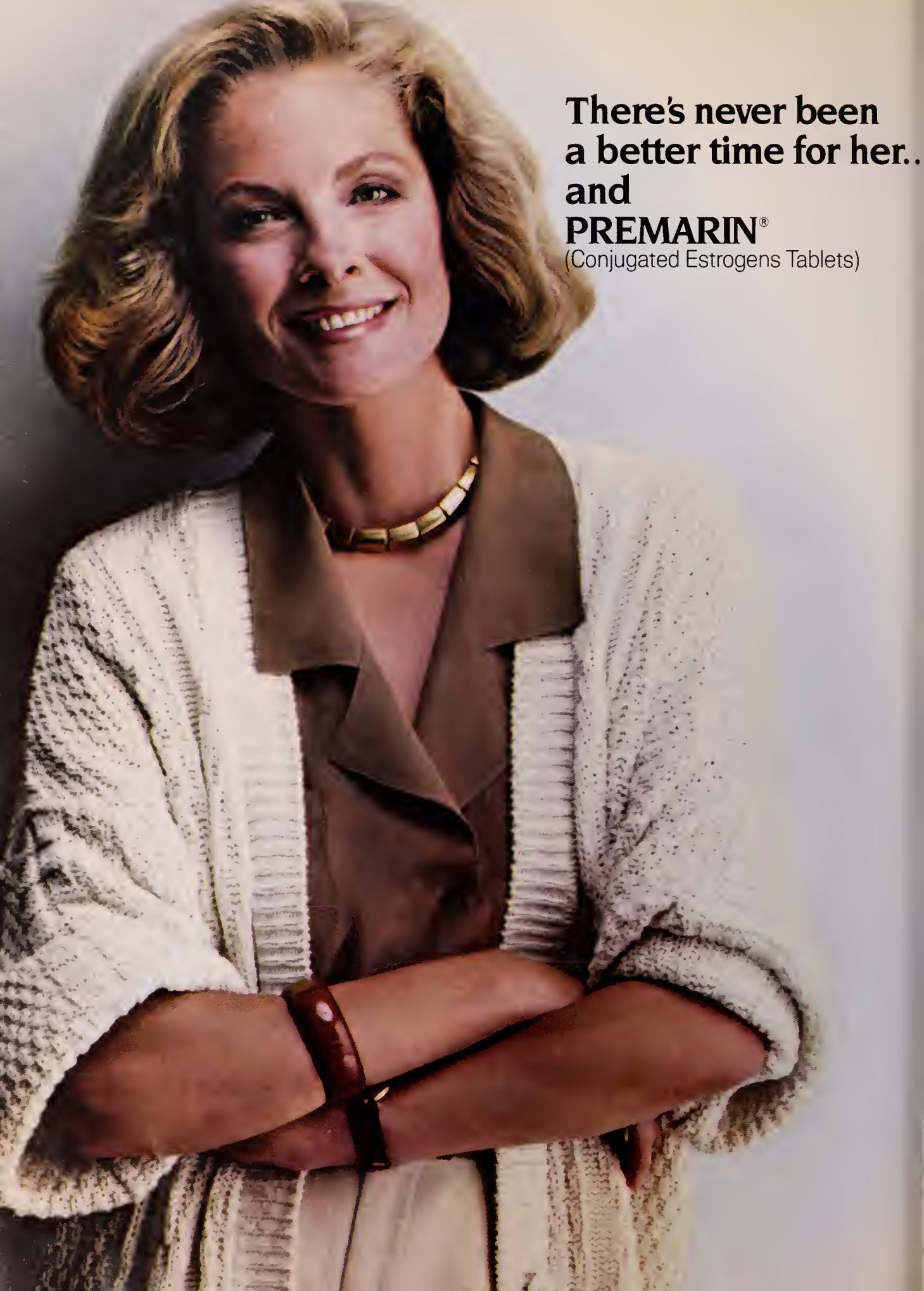
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## Low-dose control of menopausal symptoms*

PREMARIN effectively relieves vasomotor symptoms, such as hot flashes. When estrogen deficiency is limited to atrophic vaginitis, PREMARIN® (conjugated estrogens) Vaginal Cream restores the vaginal environment to its premenopausal state.

## The most widely used, most extensively studied estrogen worldwide.

# PREMARIN®

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## Most trusted for more reasons

*PREMARIN is indicated for moderate-to-severe vasomotor symptoms.

Please see following page for brief summary of prescribing information.

# For moderate-to-severe vasomotor symptoms

# PREMARIN®
## (Conjugated Estrogens Tablets)

0.3 mg 0.625 mg 0.9 mg 1.25 mg 2.5 mg

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# For atrophic vaginitis

# PREMARIN®
## (Conjugated Estrogens)

Vaginal Cream

0.625mg/g

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION AND PATIENT INFORMATION, SEE PACKAGE CIRCULARS.)*

**PREMARIN® Brand of conjugated estrogens tablets, USP**
**PREMARIN® Brand of conjugated estrogens Vaginal Cream in a nonliquefying base**

1. ESTROGENS HAVE BEEN REPORTED TO INCREASE THE RISK OF ENDOMETRIAL CARCINOMA.

Three independent case control studies have reported an increased risk of endometrial cancer in postmenopausal women exposed to exogenous estrogens for more than one year. This risk was independent of the other known risk factors for endometrial cancer. These studies are further supported by the finding that incidence rates of endometrial cancer have increased sharply since 1969 in eight different areas of the United States with population-based cancer reporting systems, an increase which may be related to the rapidly expanding use of estrogens during the last decade. The three case control studies reported that the risk of endometrial cancer in estrogen users was about 4.5 to 13.9 times greater than in nonusers. The risk appears to depend on both duration of treatment and on estrogen dose. In view of these findings, when estrogens are used for the treatment of menopausal symptoms, the lowest dose that will control symptoms should be utilized and medication should be discontinued as soon as possible. When prolonged treatment is medically indicated, the patient should be reassessed on at least a semiannual basis to determine the need for continued therapy. Although the evidence must be considered preliminary, one study suggests that cyclic administration of low doses of estrogen may carry less risk than continuous administration; it therefore appears prudent to utilize such a regimen. Close clinical surveillance of all women taking estrogens is important. In all cases of undiagnosed persistent or recurring abnormal vaginal bleeding, adequate diagnostic measures should be undertaken to rule out malignancy. There is no evidence at present that "natural" estrogens are more or less hazardous than "synthetic" estrogens at equiestrogenic doses.

2. ESTROGENS SHOULD NOT BE USED DURING PREGNANCY.
The use of female sex hormones, both estrogens and progestogens, during early pregnancy may seriously damage the offspring. It has been shown that females exposed in utero to diethylstilbestrol, a non-steroidal estrogen, have an increased risk of developing in later life a form of vaginal or cervical cancer that is ordinarily extremely rare. This risk has been estimated as not greater than 4 per 1,000 exposures. Furthermore, a high percentage of such exposed women (from 30% to 90%) have been found to have vaginal adenosis, epithelial changes of the vagina and cervix. Although these changes are histologically benign, it is not known whether they are precursors of malignancy. Although similar data are not available with the use of other estrogens, it cannot be presumed they would not induce similar changes. Several reports suggest an association between intrauterine exposure to female sex hormones and congenital anomalies, including congenital heart defects and limb reduction defects. One case control study estimated a 4.7-fold increased risk of limb reduction defects in infants exposed in utero to sex hormones (oral contraceptives, hormone withdrawal tests for pregnancy, or attempted treatment for threatened abortion). Some of these exposures were very short and involved only a few days of treatment. The data suggest that the risk of limb reduction defects in exposed fetuses is somewhat less than 1 per 1,000. In the past, female sex hormones have been used during pregnancy in an attempt to treat threatened or habitual abortion. There is considerable evidence that estrogens are ineffective for these indications, and there is no evidence from well controlled studies that progestogens are effective for these uses. If PREMARIN is used during pregnancy, or if the patient becomes pregnant while taking this drug, she should be apprised of the potential risks to the fetus, and the advisability of pregnancy continuation.

**DESCRIPTION:** PREMARIN (conjugated estrogens, USP) contains a mixture of estrogens, obtained exclusively from natural sources, blended to represent the average composition of material derived from pregnant mares' urine. It contains estrone, equilin, and 17α-dihydroequilin, together with smaller amounts of 17α-estradiol, equilenin, and 17α-dihydroequilenin as salts of their sulfate esters. Tablets are available in 0.3 mg, 0.625 mg, 0.9 mg, 1.25 mg, and 2.5 mg strengths of conjugated estrogens. Cream is available as 0.625 mg conjugated estrogens per gram.

**INDICATIONS AND USAGE:** PREMARIN (conjugated estrogens tablets, USP): Moderate-to-severe *vasomotor* symptoms associated with the menopause. (There is no evidence that estrogens are effective for nervous symptoms or depression without associated vasomotor symptoms and they should not be used to treat such conditions.) **Osteoporosis (abnormally low bone mass).** Atrophic vaginitis. Kraurosis vulvae. Female castration.

PREMARIN (conjugated estrogens) Vaginal Cream is indicated in the treatment of atrophic vaginitis and kraurosis vulvae. PREMARIN HAS NOT BEEN SHOWN TO BE EFFECTIVE FOR ANY PURPOSE DURING PREGNANCY AND ITS USE MAY CAUSE SEVERE HARM TO THE FETUS (SEE BOXED WARNING).

**Concomitant Progestin Use:** The lowest effective dose appropriate for the specific indication should be utilized. Studies of the addition of a progestin for 7 or more days of a cycle of estrogen administration have reported a lowered incidence of endometrial hyperplasia. Morphological and biochemical studies of the endometrium suggest that 10 to 13 days of progestin are needed to provide maximal maturation of the endometrium and to eliminate any hyperplastic changes. Whether this will provide protection from endometrial carcinoma has not been clearly established. There are possible additional risks which may be associated with the inclusion of progestin in estrogen replacement regimens. (See PRECAUTIONS.) The choice of progestin and dosage may be important; product labeling should be reviewed to minimize possible adverse effects.

**CONTRAINDICATIONS:** Estrogens should not be used in women (or men) with any of the following conditions: 1. Known or suspected cancer of the breast except in appropriately selected patients being treated for metastatic disease. 2. Known or suspected estrogen-dependent neoplasia. 3. Known or suspected pregnancy (See Boxed Warning). 4. Undiagnosed abnormal genital bleeding. 5. Active thrombophlebitis or thromboembolic disorders. 6. A past history of thrombophlebitis, thrombosis, or thromboembolic disorders associated with previous estrogen use (except when used in treatment of breast or prostatic malignancy).

**WARNINGS:** Long-term continuous administration of natural and synthetic estrogens in certain animal species increases the frequency of carcinomas of the breast, cervix, vagina, and liver. There are now reports that estrogens increase the risk of carcinoma of the endometrium in humans. (See Boxed Warning.) At the present time there is no satisfactory evidence that estrogens given to postmenopausal women increase the risk of cancer of the breast, although a recent study has raised this possibility. There is a need for caution in prescribing estrogens for women with a strong family history of breast cancer or who have breast nodules, fibrocystic disease, or abnormal mammograms. A recent study has reported a 2- to 3-fold increase in the risk of surgically confirmed gallbladder disease in women receiving postmenopausal estrogens.

Adverse effects of oral contraceptives may be expected at the larger doses of estrogen used to treat prostatic or breast cancer or postpartum breast engorgement; it has been shown that there is an increased risk of thrombosis in men receiving estrogens for prostatic cancer and women for postpartum breast engorgement. Users of oral contraceptives have an increased risk of diseases, such as thrombophlebitis, pulmonary embolism, stroke, and myocardial infarction. Cases of retinal thrombosis, mesenteric thrombosis, and optic neuritis have been reported in oral contraceptive users. An increased risk of postsurgery thromboembolic complications has also been reported in users of oral contraceptives. If feasible, estrogen should be discontinued at least 4 weeks before surgery of the type associated with an increased risk of thromboembolism, or during periods of prolonged immobilization. Estrogens should not be used in persons with active thrombophlebitis, thromboembolic disorders, or in persons with a history of such disorders in association with estrogen use. They should be used with caution in patients with cerebral vascular or coronary artery disease. Large doses (5 mg conjugated estrogens per day), comparable to those used to treat cancer of the prostate and breast, have been shown to increase the risk of nonfatal myocardial infarction, pulmonary embolism and thrombophlebitis. When doses of this size are used, any of the thromboembolic and thrombotic adverse effects should be considered a clear risk.

Benign hepatic adenomas should be considered in estrogen users having abdominal pain and tenderness, abdominal mass, or hypovolemic shock. Hepatocellular carcinoma has been reported in women taking estrogen-containing oral contraceptives. Increased blood pressure may occur with use of estrogens in the menopause and blood pressure should be monitored with estrogen use. A worsening of glucose tolerance has been observed in patients on estrogen-containing oral contraceptives. For this reason, diabetic patients should be carefully observed. Estrogens may lead to severe hypercalcemia in patients with breast cancer and bone metastases.

**PRECAUTIONS:** Physical examination and a complete medical and family history should be taken prior to the initiation of any estrogen therapy with special reference to blood pressure, breasts, abdomen, and pelvic organs, and should include a Papanicolaou smear. As a general rule, estrogen should not be prescribed for longer than one year without another physical examination being performed. Conditions influenced by fluid retention such as asthma, epilepsy, migraine, and cardiac or renal dysfunction, require careful observation. Certain patients may develop manifestations of excessive estrogenic stimulation, such as abnormal or excessive uterine bleeding, mastodynia, etc. Prolonged administration of unopposed estrogen therapy has been reported to increase the risk of endometrial hyperplasia in some patients. Oral contraceptives appear to be associated with an increased incidence of mental depression. Patients with a history of depression should be carefully observed. Preexisting uterine leiomyomata may increase in size during estrogen use. The pathologist should be advised of estrogen therapy when relevant specimens are submitted. If jaundice develops in any patient receiving estrogen, the medication should be discontinued while the cause is investigated. Estrogens should be used with care in patients with impaired liver function, renal insufficiency, metabolic bone diseases associated with hypercalcemia, or in young patients in whom bone growth is not complete. If concomitant progestin therapy is used, potential risks may include adverse effects on carbohydrate and lipid metabolism.

The following changes may be expected with larger doses of estrogen:

a. Increased sulfobromophthalein retention.
b. Increased prothrombin and factors VII, VIII, IX, and X; decreased antithrombin 3; increased norepinephrine-induced platelet aggregability.
c. Increased thyroid binding globulin (TBG) leading to increased circulating total thyroid hormone, as measured by PBI, T4 by column, or T4 by radioimmunoassay. Free T3 resin uptake is decreased, reflecting the elevated TBG; free T4 concentration is unaltered.
d. Impaired glucose tolerance.
e. Decreased pregnanediol excretion.
f. Reduced response to metyrapone test.
g. Reduced serum folate concentration.
h. Increased serum triglyceride and phospholipid concentration. As a general principle, the administration of any drug to nursing mothers should be done only when clearly necessary since many drugs are excreted in human milk.

**ADVERSE REACTIONS:** The following have been reported with estrogenic therapy, including oral contraceptives: breakthrough bleeding, spotting, change in menstrual flow, dysmenorrhea; premenstrual-like syndrome, amenorrhea during and after treatment; increase in size of uterine fibromyomata; vaginal candidiasis, change in cervical erosion and in degree of cervical secretion; cystitis-like syndrome; tenderness, enlargement, secretion (of breasts); nausea, vomiting, abdominal cramps, bloating; cholestatic jaundice; chloasma or melasma which may persist when drug is discontinued; erythema multiforme; erythema nodosum; hemorrhagic eruption; loss of scalp hair; hirsutism; steepening of corneal curvature; intolerance to contact lenses; headache, migraine; dizziness, mental depression, chorea; increase or decrease in weight; reduced carbohydrate tolerance; aggravation of porphyria; edema; changes in libido.

**ACUTE OVERDOSAGE:** May cause nausea, and withdrawal bleeding may occur in females.

**DOSAGE AND ADMINISTRATION:**

**PREMARIN® Brand of conjugated estrogens tablets, USP**

1. *Given cyclically for short-term use only.* For treatment of moderate to severe *vasomotor* symptoms, atrophic vaginitis, or kraurosis vulvae associated with the menopause (0.3 to 1.25 mg or more daily). The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible. Administration should be cyclic (eg, three weeks on and one week off). Attempts to discontinue or taper medication should be made at three- to six-month intervals.

2. *Given cyclically:* Female castration. Osteoporosis. Female castration—1.25 mg daily, cyclically. Adjust upward or downward according to response of the patient. For maintenance, adjust dosage to lowest level that will provide effective control. Osteoporosis—0.625 mg daily. Administration should be cyclic (eg, three weeks on and one week off).

Patients with an intact uterus should be monitored for signs of endometrial cancer and appropriate measures taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

**PREMARIN® Brand of conjugated estrogens Vaginal Cream**

*Given cyclically for short-term use only.* For treatment of atrophic vaginitis or kraurosis vulvae.

The lowest dose that will control symptoms should be chosen and medication should be discontinued as promptly as possible.

Administration should be cyclic (eg, three weeks on and one week off).

Attempts to discontinue or taper medication should be made at three-to-six month intervals.

Usual dosage range: 2 to 4 g daily, intravaginally, depending on the severity of the condition.

Treated patients with an intact uterus should be monitored closely for signs of endometrial cancer and appropriate diagnostic measures should be taken to rule out malignancy in the event of persistent or recurring abnormal vaginal bleeding.

**References:**

**1.** Whitehead MI, Townsend PT, Pryse-Davies J, et al: Effects of estrogens and progestins on the biochemistry and morphology of the postmenopausal endometrium. *N Engl J Med* 1981;305:1599-1605 **2.** Paterson MEL, Wade-Evans T, Sturdee DW, et al: Endometrial disease after treatment with oestrogens and progestogens in the climacteric. *Br Med J* 1980;280:822-824. **3.** Magos AL, Brincat M, Studd JWW, et al: Amenorrhea and endometrial atrophy with continuous oral estrogen and progestogen therapy in postmenopausal women. *Obstet Gynecol* 1985; 67: 496-499. **4.** Whitehead MI, Lane G, Siddle N, et al: Avoidance of endometrial hyperstimulation in estrogen-treated postmenopausal women. *Semin Reprod Endocrinol* 1983;1:1;41-52. **5.** Barnes RB, Roy S, Lobo RA: Comparison of lipid and androgen levels after conjugated estrogen or depo-medroxyprogesterone acetate treatment in postmenopausal women. *Obstet Gynecol* 1985;66:216-219

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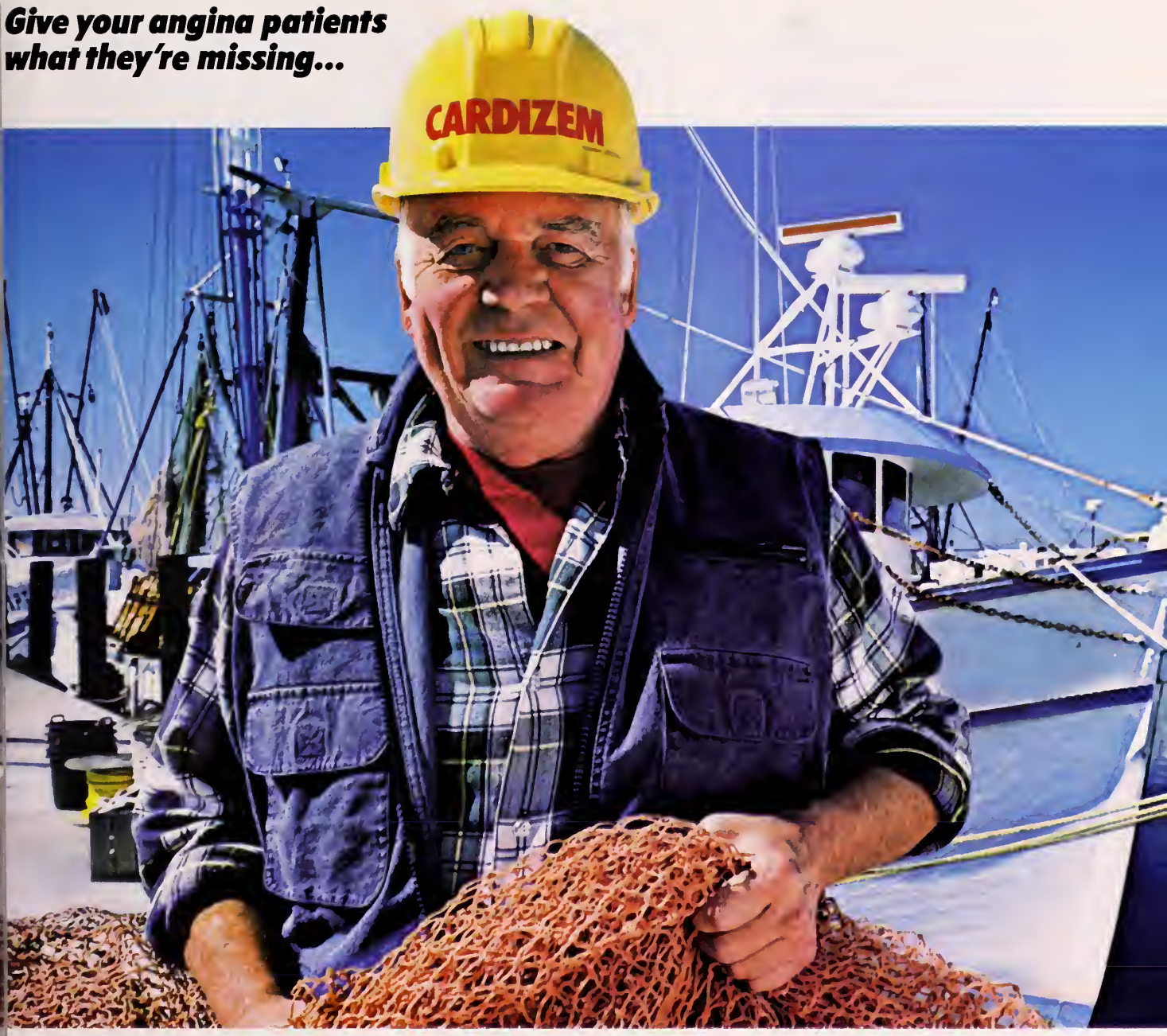


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- ***Proven efficacy when used alone in angina[1]***
- ***Compatible with other antianginals[2,3]****
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD[4,5]***

***See Warnings and Precautions.**

*Please see brief summary of prescribing information on the next page.*

# CARDIZEM® FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

## diltiazem HCl/Marion

## 60 mg tid or qid

**Brief Summary**
*Professional Use Information*

***CARDIZEM®***
*(diltiazem HCl) 30 mg and 60 mg Tablets*

***CONTRAINDICATIONS***

*CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).*

***WARNINGS***

1. ***Cardiac Conduction.*** *CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.*
2. ***Congestive Heart Failure.*** *Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).*
   *Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.*
3. ***Hypotension.*** *Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.*
4. ***Acute Hepatic Injury.*** *In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)*

***PRECAUTIONS***

***General.*** *CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes, however, these changes were reversible with continued dosing.*

***Drug Interaction.*** *Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)*

*Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.*

***Carcinogenesis, Mutagenesis, Impairment of Fertility.*** *A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.*

***Pregnancy.*** *Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.*

*There are no well-controlled studies in pregnant women, therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.*

***Nursing Mothers.*** *Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.*

***Pediatric Use.*** *Safety and effectiveness in children have not been established.*

***ADVERSE REACTIONS***

*Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.*

*In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.*

*The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):*

| | |
|---|---|
| *Cardiovascular:* | *Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope.* |
| *Nervous System:* | *Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor.* |
| *Gastrointestinal:* | *Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase.* |
| *Dermatologic:* | *Petechiae, pruritus, photosensitivity, urticaria.* |
| *Other:* | *Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties.* |

*The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.* ***Issued 7/86***

*See complete Professional Use Information before prescribing.*

***References: 1.*** *Pepine CJ, Feldman RL, Hill JA, et al. Clinical outcome after treatment of rest angina with calcium blockers. Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J. 1983, 106(6):1341-1347.* ***2.*** *Shapiro W. Calcium channel blockers. Actions on the heart and uses in ischemic heart disease. Consultant 1984;24(Dec):150-159.* ***3.*** *Johnston DL, Lesaway R, Humen DP, et al. Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol. 1985;55:680-687.* ***4.*** *Cohn PF, Braunwald E. Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease. A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap. 39.* ***5.*** *Schroeder JS: Calcium and beta blockers in ischemic heart disease. When to use which. Mod Med 1982;50(Sept):94-116.*

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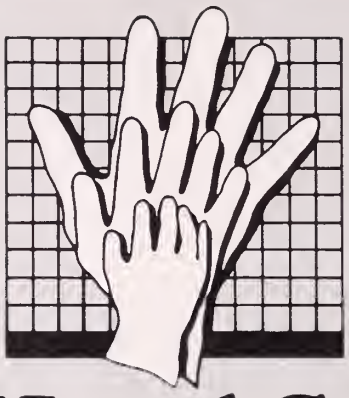

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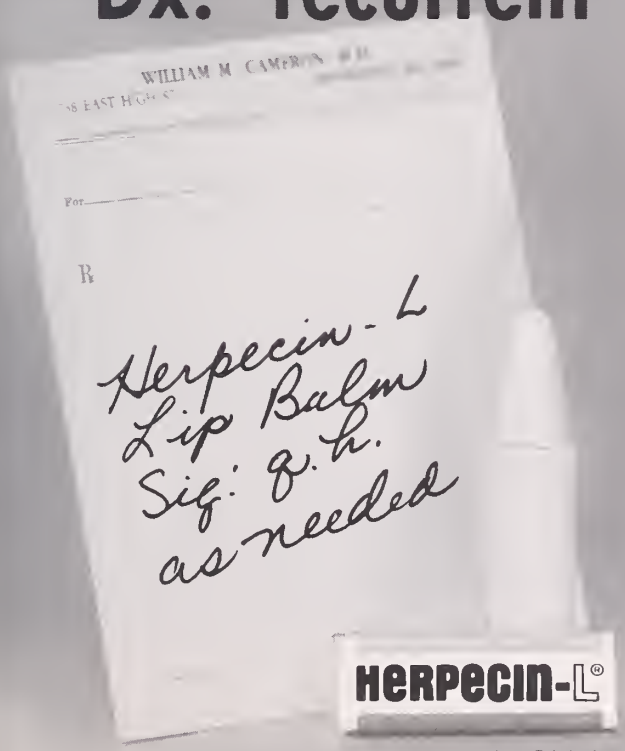


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Articles submitted for publication, including Annual Meeting papers, become the sole property of the JOURNAL and must not have been published elsewhere. The Editorial Board reserves the right to edit any material submitted. Manuscripts must be typewritten, double-spaced, and submitted in duplicate. Receipt of manuscripts will be acknowledged, and unpublished manuscripts will be returned. The JOURNAL does not assume responsibility for the statements or opinions of any contributor.

**Style**

All manuscripts should adhere to the style adopted by the American Medical Association as illustrated in *JAMA* and detailed in the AMA's *Manual for Authors & Editors*. Footnotes, bibliographies, and legends for illustrations should be typewritten, double-spaced, on separate sheets. References are to be listed in the order of their appearance in the article.

**Illustrations**

Illustrations other than the author's will not be accepted for publication unless accompanied by written permission from the original source. Illustrations should be labeled with the author's name and must be numbered in the order in which they are referred to in the article. The quality of all illustrations must be in keeping with the quality of the magazine.

**News**

Readers are encouraged to submit news items of interest to Oklahoma physicians. Where dates of meetings, etc, are important, please remember that each issue closes on the first day of the *preceding* month and reaches subscribers in the latter half of the month of publication.

**Reprints**

Authors will receive reprint order forms from the Transcript Press, 222 East Eufaula, Norman, Oklahoma 73069, prior to publication of their articles. Other requests for reprints must be made to the Transcript Press within 30 days after publication.

**Back Issues**

Microfilm copies of back issues of the JOURNAL can be purchased from University Microfilms International, 300 North Zeeb Road, Ann Arbor, Michigan 48106.

# Convention '87 — Moving Forward Together

OSMA and OSMAA are "MOVING FORWARD TOGETHER" toward Convention '87!! In an effort to recognize and encourage a spirit of cooperation, mutual interest, and unity among physicians and physician spouses, OSMAA has chosen "MOVING FORWARD TOGETHER" as its theme for the 1987 Annual Meeting. All meetings and events will be held May 1-3, 1987, at the Sheraton Century Center Hotel, Oklahoma City.

For the first time, in 1987, all social events, including luncheons, parties, and Sunday morning breakfast, will be held in conjunction with physicians. You can look forward to a "come and go" soup, salad, and sandwich luncheon upon arrival Friday afternoon; reception and dinner Friday night hosted by the OU Alumni Association; luncheon Saturday featuring Dr William S. Hotchkiss, president-elect of the AMA, as speaker; and the traditional President's Inaugural Dinner-Dance Saturday evening. In addition, to celebrate the running of the Kentucky Derby Saturday afternoon, OSMA will host its own "Derby Day Party." The OSMA Convention Program will include topics that are of interest to both physicians and spouses, and auxilians are encouraged to attend.

## Image Professionals

Auxiliary is pleased this year to have Gale Bollinger and Joy Richardson, IMAGE PROFESSIONALS, available for consultation. Image Professionals helps women improve their self-image through personal coloring, practical wardrobing, proper skin care, and make-up techniques. Both Gale and Joy have degrees from the University of Oklahoma in fashion-related fields, over 20 years of modeling experience, and are certified graduates of the d'Saison School of Color Analysis and Cosmetic Training. They will be available for private consultation by appointment Friday afternoon, May 1, and Saturday morning, May 2, at a special cost to auxilians of $25.

## Hospitality and Health Education

The Auxiliary Hospitality Room will be a center of activity, with on-going health education films and exhibits, the AMA-ERF Silent Auction, featuring hand-made items donated by each county, and refreshments. It will be open Friday afternoon and Saturday for meeting friends, learning about various health-related topics, and relaxing.

## Visiting Dignitaries

Auxiliary is honored to have as guests for Convention '87 Jean Hill (Mrs J. Edward), southern regional vice-president, AMAA, and Virginia Hopper (Mrs John M.), president, Southern Medical Association Auxiliary.

Mrs Hill is from Hollandale, Mississippi, and is serving her second term as southern regional vice-president. She has previously served at the national level in numerous positions including health projects chairman and member of the membership committee. She has served on the state level as president, treasurer, and member of the Membership and Finance committees. She has served her county as president, and chairman for both AMA-ERF and Doctor's Day. She and Dr Hill, a family practitioner, have two daughters.

Mrs Hopper is from Baton Rouge, Louisiana, and has served in the Southern Medical Association Auxiliary as state councilor, memorial chairman, Doctor's Day judges chairman, historian, membership chairman, regional vice-president, and president-elect. She has been president of both her local and state auxiliaries, is a member of the Junior League of Baton Rouge, and has worked extensively in her church and children's schools. She and Dr Hopper, a neurologist, have four children and four grandchildren.

Convention '87 promises to be an informative, entertaining, and FUN three days! Mark your calendars, watch for registration materials, and plan to join OSMA and OSMAA "MOVING FORWARD TOGETHER" May 1-3 in Oklahoma City.

*—Kathy Woodall*
*OSMAA Convention Chairman*

■ **The American Board of Medical Specialties** has produced a 24-page booklet explaining medical specialty areas for the lay public. Multiple copies of the booklet will be available to individuals or organizations for 25 cents per copy. Persons interested in distributing the publication should write to the American Board of Medical Specialties, One American Plaza, Suite 805, Evanston, IL 60201.

■ **Physicians should play a key role in efforts** to reduce the safety threat that guns in the home pose to children, says a report in February's *American Journal of Diseases of Children (AJDC)*. Patti J. Patterson, MD, and Leigh R. Smith, MD, of the University of Texas Medical Branch (UTMB), Galveston, say doctors — especially pediatricians — have successfully promoted child safety laws for automobiles, poisons, and flammable fabrics; "a similar effort should be made with regard to firearms." Patterson and Smith surveyed 150 families from the UTMB pediatric outpatient clinic and found that 38% kept at least one gun in the home; more than half this group said the gun was loaded at all times and 10% said it was loaded, unlocked, and within a child's reach. Despite a limited study sample, "the overall importance of household firearms as a threat to safety should not be overlooked," the authors say.

■ **The increasing popularity of faculty practice** plans — income-generating, academic-based clinical practice groups — may create a "town/gown" split with competing community physicians, a report in the *Journal of the American Medical Association* suggests. Gordon MacLeod, MD, of the University of Pittsburgh, and colleagues assessed the attitudes of academic and community health leaders toward faculty practice plans in a sample of seven university communities. While it was agreed that the community benefits from these plans, the potential rift was evident from a number of community responses. Community health leaders objected to the plans' tax-exempt status; doubted medical schools can compete successfully with community practice; feared resulting reduced income; agreed competition adversely affects cooperation in the medical community at large; and appeared to envy the ability of faculty physicians to buffer their incomes by teaching and research.

■ **Free subscriptions to *Alcoholism & Addiction Magazine*** are being offered to physicians until April 30, 1987. Licensed physicians should send their requests on their professional letterhead to *Alcoholism & Addiction Magazine*, 23860 Miles Road, Cleveland, OH 44128.

■ **Three Tulsa physicians have been named as** 1987 Doctors of the Year by the Tulsa County Medical Society (TCMS) Auxiliary. Recipients of the awards are George W. Prothro, MD, a public health physician; Harlan Thomas, MD, a retired family practitioner; and Lynwood Heaver, MD, a retired psychiatrist. All are Life Members of TCMS. Presentation ceremonies were held February 8 in conjunction with the inauguration of TCMS president Jerry L. Puls, MD.

■ **Tulsa physicians have been busy writing** as well as practicing medicine. "Pharmacokinetics and Nephrotoxicity of Gentamicin in LBW Infants," by George P. Giacoia, MD, a pediatrician, was published in a recent issue of the *Journal of Pediatrics*. Patrick D. Lester, MD, et al wrote "Ferromagnetic Screening Around a Superconducting Magnetic Resonance Imager," which appeared in the October issue of *Health Physics*.

■ **The CME course "Current Trends in Obstetrics and Gynecology"** will be offered Wednesday through Friday, May 27-29, by the University of Oklahoma College of Medicine. For information contact Magdalen De Bault, associate director, CME, OU College of Medicine, PO Box 26901, Room 164E, LB, Oklahoma City, OK 73190.

■ **A one-day conference, "Cleft Palate—** Disordered Speech, Videofluoroscopy, Surgical Repair," will be presented at the Oklahoma Center for Continuing Education on Saturday, May 9, from 9 am to 2 pm. Speakers will be Hans Norberg, MD, a plastic surgeon in private practice in Tulsa, and Robert Thompson, PhD, director of speech and audiology at Children's Medical Center, Tulsa. For information contact Cindy Jolls, Oklahoma Center for Continuing Education, 405-325-7378; Dr Norberg, 918-749-3466; or Dr Thompson, 918-664-6600.

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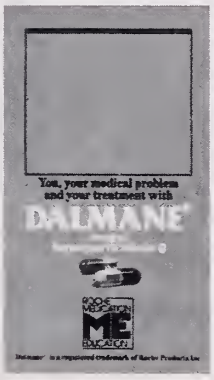

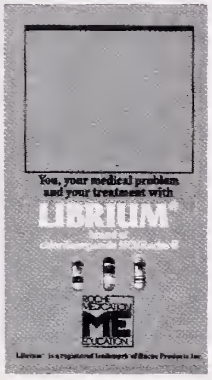

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**Contraindications:** Known hypersensitivity to the drug.
**Warnings:** Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
**Precautions:** In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (e.g., excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.
**Adverse Reactions:** Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
**Usual Daily Dosage:** Individualize for maximum beneficial effects. *Oral—Adults.* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)
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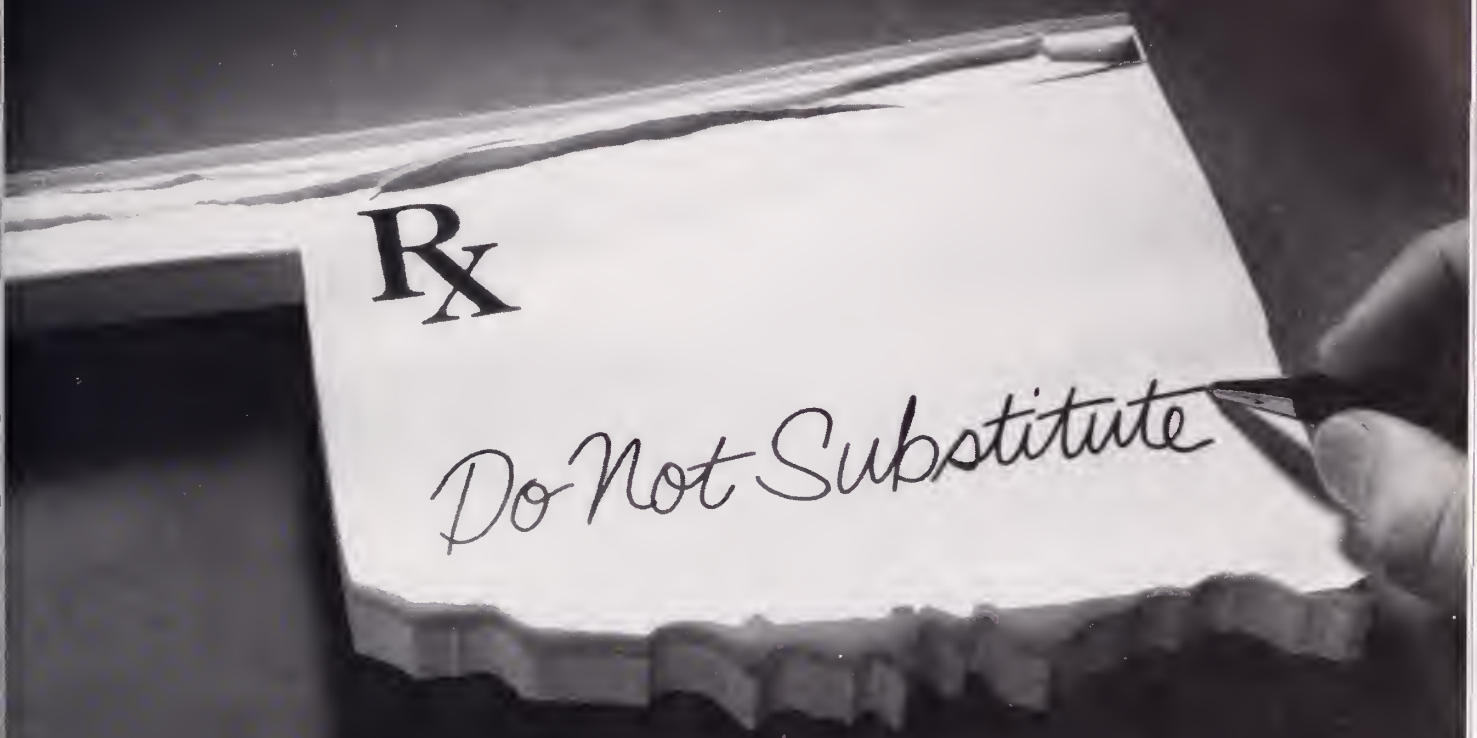


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The change making seminar attendance mandatory was implemented by the PLICO Board of Directors in late 1985, and a special endorsement outlining the requirement was included in all 1986 PLICO professional liability insurance policies. Any physician needing to attend in 1987, and failing to do so, will not be eligible for renewal of his or her PLICO professional liability insurance for calendar year 1988.

A Seminar Registration Form is located at the bottom of this page and a schedule of upcoming seminars is included. The registration form may be used for any seminar, but please **specify the date** you would like to attend. It is advisable to pre-register for the seminar you would like to attend.

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## 1987 LOSS PREVENTION SEMINARS

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| June 6 | Sat. 2-5 p.m. | TULSA<br>Marriott |
| July 18 | Sat. 2-5 p.m. | LAWTON<br>Holiday Inn |
| August 8 | Sat. 2-5 p.m. | WOODWARD<br>Park Inn |
| September 19 | Sat. 8:30 a.m.-11:30 a.m. | McALESTER<br>Holiday Inn |
| September 26 | Sat. 8:30 a.m.-11:30 a.m. | OKLAHOMA CITY<br>Lincoln Plaza |

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Please register the following physician to attend the 1987 Loss Prevention Seminar indicated below (PLEASE TYPE OR PRINT):

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LONG ACTING CAPSULES

Like conventional INDERAL Tablets, INDERAL LA should not be used in the presence of congestive heart failure, sinus bradycardia, cardiogenic shock, heart block greater than first degree, and bronchial asthma.

*After a 30-day trial with INDERAL LA, physicians reported that 90% of the patients would remain on INDERAL LA.

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INDERAL LA 60 mg
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Please see next page for brief summary of prescribing information

# The one you know best keeps looking better

BRIEF SUMMARY *(FOR FULL PRESCRIBING INFORMATION, SEE PACKAGE CIRCULAR.)*

**INDERAL® LA** brand of propranolol hydrochloride **(Long Acting Capsules)**

**DESCRIPTION.** INDERAL LA is formulated to provide a sustained release of propranolol hydrochloride. INDERAL LA is available as 60 mg, 80 mg, 120 mg, and 160 mg capsules.

**CLINICAL PHARMACOLOGY.** INDERAL is a nonselective, beta-adrenergic receptor-blocking agent possessing no other autonomic nervous system activity. It specifically competes with beta-adrenergic receptor-stimulating agents for available receptor sites. When access to beta-receptor sites is blocked by INDERAL, the chronotropic, inotropic, and vasodilator responses to beta-adrenergic stimulation are decreased proportionately.

INDERAL LA Capsules (60, 80, 120, and 160 mg) release propranolol HCl at a controlled and predictable rate. Peak blood levels following dosing with INDERAL LA occur at about 6 hours and the apparent plasma half-life is about 10 hours. When measured at steady state over a 24-hour period the areas under the propranolol plasma concentration-time curve (AUCs) for the capsules are approximately 60% to 65% of the AUCs for a comparable divided daily dose of INDERAL Tablets. The lower AUCs for the capsules are due to greater hepatic metabolism of propranolol, resulting from the slower rate of absorption of propranolol. Over a twenty-four (24) hour period, blood levels are fairly constant for about twelve (12) hours then decline exponentially.

INDERAL LA should not be considered a simple mg-for-mg substitute for conventional propranolol and the blood levels achieved do not match (are lower than) those of two to four times daily dosing with the same dose. When changing to INDERAL LA from conventional propranolol, a possible need for retitration upwards should be considered especially to maintain effectiveness at the end of the dosing interval. In most clinical settings, however, such as hypertension or angina where there is little correlation between plasma levels and clinical effect, INDERAL LA has been therapeutically equivalent to the same mg dose of conventional INDERAL as assessed by 24-hour effects on blood pressure and on 24-hour exercise responses of heart rate, systolic pressure and rate pressure product. INDERAL LA can provide effective beta blockade for a 24-hour period.

**INDICATIONS AND USAGE. Hypertension:** INDERAL LA is indicated in the management of hypertension; it may be used alone or used in combination with other antihypertensive agents, particularly a thiazide diuretic. INDERAL LA is not indicated in the management of hypertensive emergencies.

**Angina Pectoris Due to Coronary Atherosclerosis:** INDERAL LA is indicated for the long-term management of patients with angina pectoris.

**Migraine:** INDERAL LA is indicated for the prophylaxis of common migraine headache. The efficacy of propranolol in the treatment of a migraine attack that has started has not been established and propranolol is not indicated for such use.

**Hypertrophic Subaortic Stenosis:** INDERAL LA is useful in the management of hypertrophic subaortic stenosis, especially for treatment of exertional or other stress-induced angina, palpitations, and syncope. INDERAL LA also improves exercise performance. The effectiveness of propranolol hydrochloride in this disease appears to be due to a reduction of the elevated outflow pressure gradient which is exacerbated by beta-receptor stimulation. Clinical improvement may be temporary.

**CONTRAINDICATIONS.** INDERAL is contraindicated in 1) cardiogenic shock, 2) sinus bradycardia and greater than first-degree block, 3) bronchial asthma, 4) congestive heart failure (see WARNINGS) unless the failure is secondary to a tachyarrhythmia treatable with INDERAL.

**WARNINGS.** CARDIAC FAILURE. Sympathetic stimulation may be a vital component supporting circulatory function in patients with congestive heart failure, and its inhibition by beta blockade may precipitate more severe failure. Although beta blockers should be avoided in overt congestive heart failure, if necessary, they can be used with close follow-up in patients with a history of failure who are well compensated and are receiving digitalis and diuretics. Beta-adrenergic blocking agents do not abolish the inotropic action of digitalis on heart muscle.

IN PATIENTS WITHOUT A HISTORY OF HEART FAILURE, continued use of beta blockers can, in some cases, lead to cardiac failure. Therefore, at the first sign or symptom of heart failure, the patient should be digitalized and/or treated with diuretics, and the response observed closely, or INDERAL should be discontinued (gradually, if possible).

IN PATIENTS WITH ANGINA PECTORIS, there have been reports of exacerbation of angina and, in some cases, myocardial infarction, following *abrupt* discontinuance of INDERAL therapy. Therefore, when discontinuance of INDERAL is planned, the dosage should be gradually reduced over at least a few weeks, and the patient should be cautioned against interruption or cessation of therapy without the physician's advice. If INDERAL therapy is interrupted and exacerbation of angina occurs, it usually is advisable to reinstitute INDERAL therapy and take other measures appropriate for the management of unstable angina pectoris. Since coronary artery disease may be unrecognized, it may be prudent to follow the above advice in patients considered at risk of having occult atherosclerotic heart disease who are given propranolol for other indications.

**Nonallergic Bronchospasm (eg, chronic bronchitis, emphysema)** —PATIENTS WITH BRONCHOSPASTIC DISEASES SHOULD IN GENERAL NOT RECEIVE BETA BLOCKERS. INDERAL should be administered with caution since it may block bronchodilation produced by endogenous and exogenous catecholamine stimulation of beta receptors.

MAJOR SURGERY. The necessity or desirability of withdrawal of beta-blocking therapy prior to major surgery is controversial. It should be noted, however, that the impaired ability of the heart to respond to reflex adrenergic stimuli may augment the risks of general anesthesia and surgical procedures.

INDERAL (propranolol HCl), like other beta blockers, is a competitive inhibitor of beta-receptor agonists and its effects can be reversed by administration of such agents, eg, dobutamine or isoproterenol. However, such patients may be subject to protracted severe hypotension. Difficulty in starting and maintaining the heartbeat has also been reported with beta blockers.

DIABETES AND HYPOGLYCEMIA. Beta blockers should be used with caution in diabetic patients if a beta-blocking agent is required. Beta blockers may mask tachycardia occurring with hypoglycemia, but other manifestations such as dizziness and sweating may not be significantly affected. Following insulin-induced hypoglycemia, propranolol may cause a delay in the recovery of blood glucose to normal levels.

THYROTOXICOSIS. Beta blockade may mask certain clinical signs of hyperthyroidism. Therefore, abrupt withdrawal of propranolol may be followed by an exacerbation of symptoms of hyperthyroidism, including thyroid storm. Propranolol may change thyroid function tests, increasing $T_4$ and reverse $T_3$, and decreasing $T_3$.

IN PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME, several cases have been reported in which, after propranolol, the tachycardia was replaced by a severe bradycardia requiring a demand pacemaker. In one case, this resulted after an initial dose of 5 mg propranolol.

**PRECAUTIONS.** GENERAL. Propranolol should be used with caution in patients with impaired hepatic or renal function. INDERAL (propranolol HCl) is not indicated for the treatment of hypertensive emergencies.

Beta-adrenoreceptor blockade can cause reduction of intraocular pressure. Patients should be told that INDERAL may interfere with the glaucoma screening test. Withdrawal may lead to a return of increased intraocular pressure.

CLINICAL LABORATORY TESTS. Elevated blood urea levels in patients with severe heart disease, elevated serum transaminase, alkaline phosphatase, lactate dehydrogenase.

DRUG INTERACTIONS. Patients receiving catecholamine-depleting drugs such as reserpine should be closely observed if INDERAL is administered. The added catecholamine-blocking action may produce an excessive reduction of resting sympathetic nervous activity which may result in hypotension, marked bradycardia, vertigo, syncopal attacks, or orthostatic hypotension.

Caution should be exercised when patients receiving a beta blocker are administered a calcium-channel-blocking drug, especially intravenous verapamil, for both agents may depress myocardial contractility or atrioventricular conduction. On rare occasions, the concomitant intravenous use of a beta blocker and verapamil has resulted in serious adverse reactions, especially in patients with severe cardiomyopathy, congestive heart failure or recent myocardial infarction.

*Aluminum hydroxide gel* greatly reduces intestinal absorption of propranolol.

*Ethanol* slows the rate of absorption of propranolol.

*Phenytoin, phenobarbitone,* and *rifampin* accelerate propranolol clearance.

*Chlorpromazine,* when used concomitantly with propranolol, results in increased plasma levels of both drugs.

*Antipyrine* and *lidocaine* have reduced clearance when used concomitantly with propranolol.

*Thyroxine* may result in a lower than expected $T_3$ concentration when used concomitantly with propranolol.

*Cimetidine* decreases the hepatic metabolism of propranolol, delaying elimination and increasing blood levels.

*Theophylline* clearance is reduced when used concomitantly with propranolol.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY. Long-term studies in animals have been conducted to evaluate toxic effects and carcinogenic potential. In 18-month studies in both rats and mice, employing doses up to 150 mg/kg/day, there was no evidence of significant drug-induced toxicity. There were no drug-related tumorigenic effects at any of the dosage levels. Reproductive studies in animals did not show any impairment of fertility that was attributable to the drug.

PREGNANCY: Pregnancy Category C. INDERAL has been shown to be embryotoxic in animal studies at doses about 10 times greater than the maximum recommended human dose.

There are no adequate and well-controlled studies in pregnant women. INDERAL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

NURSING MOTHERS. INDERAL is excreted in human milk. Caution should be exercised when INDERAL (propranolol HCl) is administered to a nursing woman.

PEDIATRIC USE. Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS.** Most adverse effects have been mild and transient and have rarely required the withdrawal of therapy.

*Cardiovascular.* Bradycardia, congestive heart failure, intensification of AV block, hypotension, paresthesia of hands, thrombocytopenic purpura, arterial insufficiency, usually of the Raynaud type.

*Central Nervous System.* Light-headedness, mental depression manifested by insomnia, lassitude, weakness, fatigue, reversible mental depression progressing to catatonia, visual disturbances, hallucinations, vivid dreams, an acute reversible syndrome characterized by disorientation for time and place, short-term memory loss, emotional lability, slightly clouded sensorium, and decreased performance on neuropsychometrics. For immediate formulations, fatigue, lethargy and vivid dreams appear dose related.

*Gastrointestinal:* Nausea, vomiting, epigastric distress, abdominal cramping, diarrhea, constipation, mesenteric arterial thrombosis, ischemic colitis.

*Allergic.* Pharyngitis and agranulocytosis, erythematous rash, fever combined with aching and sore throat, laryngospasm and respiratory distress.

*Respiratory.* Bronchospasm.

*Hematologic.* Agranulocytosis, nonthrombocytopenic purpura, thrombocytopenic purpura.

*Auto-Immune.* In extremely rare instances, systemic lupus erythematosus has been reported.

*Miscellaneous.* Alopecia, LE-like reactions, psoriasiform rashes, dry eyes, male impotence and Peyronie's disease have been reported rarely. Oculomucocutaneous reactions involving the skin, serous membranes and conjunctivae reported for a beta blocker (practolol) have not been associated with propranolol.

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**REFERENCES:**

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| OXYCODONE | XX | XX | XX | XX | XX |

**Blank space indicates that no such activity has been reported.**

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

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Revised, April 1982. S68S


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# Meditations on Mail Order Medicine

It was a simple request. Would I please write a prescription for aspirin tablets? A ninety day supply with renewals sufficient to provide a year of therapy.

Simple enough, right?

Let's see. The request was made with a call to my office from my patient. I see him by appointment about every six months. He has hypertension and has been a patient of mine for more than twenty years. Five years ago I advised him to take one aspirin tablet daily, explaining how it might decrease his risk of suffering a heart attack or stroke.

When he made the request, my patient explained that he planned to send the prescription to a mail order drug company and that his insurance would pay for it.

As it happens, the patient who made this request is the owner of a successful, local business. He is wealthy. He owns a large home. He drives a Mercedes and his wife drives a Cadillac. Over the age of 65 years, he has made no plans to retire. He and his business are thriving, thanks to local patronage.

Although not unique, I will admit the anecdote cited is extreme. In the past six months I have written a number of prescriptions for patients who requested them in order to "send them off" and/or because their "insurance will pay for them."

Privately, my response to such requests is ambivalent. I sympathize with patients whose medication costs are burdensome at best, impoverishing at worst. Some relief, almost, irrespective of the source, is welcome and seemingly justified. However, I am forced to wonder, what is the true cost of the apparent saving?

Am I entitled to charge a fee and can I expect reimbursement for my receptionist's time and my time and my liability and my mailing expense in responding to such requests? If so, what code applies and, in filing a claim, can I add the costs involved in filing the claim? Or is this another gratuity pirated from my resources?

What are the total mailing costs of such transactions? And what are the risks of the prescriptions or the medications being lost in the mails? In the event of such loss, will I be expected to provide additional prescriptions? If so, what proof of loss shall I demand?

What are the costs of possible errors in such transactions? How about unacceptable substitutions? Generics of unidentified origin and manufacture? Uncontrolled temperatures to which temperature-sensitive drugs are exposed during shipment? Broken or mysteriously opened containers? Expiration dates not adequate to cover the period of projected use?

Finally, how much will it cost us and our patients to eliminate local pharmacies? To be unable to obtain medications that are needed immediately? To lose local accountability and responsibility? To function without a local pharmacist who protects us and our patients against inadvertent errors in writing prescriptions and who is ready and willing to answer our questions and those of our patients?

Personally, I'm not ready to provide pharmacy services to my patients or my community. Neither am I willing to carry an inventory of drugs in my office. Furthermore, I can't think of a single contribution a mail order business in New Jersey or Chicago has made to my community.

Mark it down as another piece of fiction and deception promoted by insurance companies. *They* don't pay for the savings in the cost of our patients' aspirin tablets. *We* do.

—*MRJ*

## President's Page

It is with a great sense of pride and trepidation that I assume the responsibility of the presidency of the Oklahoma State Medical Association.

I appreciate the honor you have bestowed upon me, and I recognize the challenge of following in the footsteps of the capable leadership we have had in the past. This will not be easy. Problems in the past that seemed to have simple solutions have become very complex and require complex solutions. However, when analyzed, some may have more simple solutions than we think.

Organized Medicine, physicians, and most of all, our patients, have suffered greatly over the past 22 years. What was touted and represented as the panacea for the care of our senior citizens has become a nightmare of a bureaucratic maze that now costs much much more than even the most brazen dared predict in 1963. The elderly, who were to be the beneficiaries of this Great Society, find themselves paying more out of their pockets than they did at the inception of the Medicare program, while the costs have skyrocketed.

Although Organized Medicine warned of the many dangers ahead, most of us joined in the effort to make it work. The third party payors and the money brokers have gotten into the act with corresponding increases in the cost (for each must have his profit), and we and our patients have lost our rights to contract together for the provision of the greatest medical care in the world. Now we must call an insurance technician for permission for all but the simplest of care for our patients.

The money brokers have managed to get control of medicine, ostensibly to provide their customers (our patients) with cost-effective medical care, when in reality their goal is to control the finances of medical care, and they cannot do this without the control of the deliverer of that care, the PHYSICIAN.

When was the last time you saw a reduction in third-party premium rates without an even greater reduction in benefits? Where were the money managers prior to 1965, when most physicians were taking care of many of their patients for whatever they could afford (many times this was nothing).

We must remember this: *Anything the third parties or the money brokers do is in the interests of more profits for their stockholders.* (Is Santa Claus dead?) I can hear them now: "But we are non-profit." Ain't accounting wonderful?

Now, we can all look back and see the forks in the road, the roads that we should have taken, and we have had leaders who have tried to lead us down those roads. Why did we not heed their cry? Is it because we have lost our constituency (our patients)?

Is it because of greed? Is it because we "are a castle within ourselves"? *Or is it because of lack of unity?* I suppose that one of the things that stands in the way of unity is one of the attributes that one must have to become a physician: the ability to make life and death decisions in a very lonely atmosphere in the middle of the night. Perhaps we do become a castle within ourselves? Perhaps, in individual cases, any or all of the above might be true.

*Why all the historic diatribe above?*

Because I am firmly convinced that in the very near future we will have another fork in the road where we must choose if we are going to maintain any freedom at all and if we are going to protect our patients and ourselves from the scourges of the parasites.

As many of our colleagues have urged, we have joined, we have attempted to "work within," and the result has been the lending of credibility, many times, to bad causes.

I do not know when we will make the decision that *enough is enough.* Is it now, when the anesthesiologists, pathologists, and radiologists are under siege? We didn't make that decision when the pathologists were under attack in 1978. They were subdued — but they were only a small group. (Divide and conquer?) Now, with that success under its belt, the Office of Management and Budget has three of our groups under attack. When will we realize that there, but for the grace of God, go we? When will we realize that we *all* are next?

I do not know, but of one thing I am firmly convinced — *We will do nothing without unity!*

*Enough is enough!*

M. Joe Crosthwait, MD

# Infantile Autism Reconsidered

JAMES R. ALLEN, MD

**During the past two decades, there has been a major shift in our thinking about infantile autism. The once-prevalent view that it was a psychogenic psychosis has been replaced by the view that it is a developmental disorder. There now seems to be general agreement that social relatedness and communication are deviant, not just delayed. However, disagreements over syndrome definition and diagnosis have impeded research efforts and the development of proven treatment regimens.**

During the past twenty years, there has been a radical shift in our thinking about infantile autism. The once-prevalent view that it was a psychogenic psychosis has been replaced by the view that it is a developmental disorder — a perspective typified by the title change of the *Journal of Autism and Childhood Schizophrenia* to the *Journal of Autism and Developmental Disorders*. This article focuses on some of these changes and their relevance for treatment.

## Definition

In 1938, Leo Kanner, then chief of child psychiatry at Johns Hopkins, saw a five-year-old with a cluster of symptoms which had never been described before. By 1943, he had seen ten more children who presented with a similar picture and published his now-classic article on the eleven.[1] Although he clearly appreciated the uniqueness of the syndrome, Kanner could hardly have foreseen its impact in the psychiatric world or the controveries which would surround it.

Few conditions have carried more different names than the subcategories of childhood psychoses, but no clear differences between them had been convincingly demonstrated. By the seventies, it had become evident that it was necessary to differentiate between severe mental illnesses arising during infancy and the psychoses of later childhood. The former group basically are composed of serious abnormalities in development itself and bear little relationship to the psychotic disorders of adult life. In *DSM III*,[3] *childhood psychoses* does not appear at all, nor does *childhood schizophrenia,* although schizophrenia in childhood can be diagnosed if criteria for the adult disorder are satisfied. In this confusing milieu, Kanner's syndrome of infantile autism seems to stand out.

The description of the pervasive developmental disorders which begins the section in *DSM III*[3] reads:

"The disorders in this subclass are characterized by distortions in the development of multiple basic functions that are involved in the development of social skills and language, such as attention, perception, reality testing and motor movement."

The category includes infantile autism, pervasive developmental disorder, childhood onset, which differs from the former primarily in age of onset and

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severity, and atypical pervasive developmental disorder, which is a category for similar disorders not covered by the aforementioned categories. The defining features of infantile autism are a lack of responsiveness to other people (autism), gross impairment in communicative skills, and bizarre responses to various aspects of the environment — all developing within the first 30 months of age.

Although autism is a syndrome in which certain symptoms and signs cluster together in a predictable way, there is, in reality, considerable variation in the clinical pictures of these children. It seems obvious that there are distortions in many basic psychological functions, such as attention and perception. The distortion in "reality testing" lacks theoretical and research validation. The evidence of "motor impairment" is somewhat equivocal, for many of these children do have bizarre motor movements of a functional nature and some show dyspraxia.

**Recent definitions of autism assume the disorder occurs at all levels of intelligence.**

Kanner described five symptoms as delineating the syndrome. First is the inability of the child to relate normally from the beginning of life; these children maintain what Kanner called "autistic aloneness," forming no apparent attachments to people and seeming to live as if others did not exist. Second is their failure to use language for the purpose of communication. The third is their obsessive need to maintain a sameness of the environment. Fourth is their fascination with objects, and their ability to handle them. The fifth is their good cognitive potential, as inferred from skills in isolated areas, general attractiveness, and serious facial expressions.

Over the years, this description has generally held up, with the exception of "good cognitive potential." Recent definitions of autism, such as those of *DSM III*[3] and the National Association of Autistic Children,[2] assume the disorder occurs at all levels of intelligence, with and without neurological impairment. This is a marked change from the time when clinicians generally believed that the presence of neurological signs or of mental retardation ruled out the diagnosis, even if the child met the behavioral criteria, or when we spoke of "primary" and "secondary" early infantile autism. However, there is growing recognition that those with normal and below-normal IQs seem to represent very different subgroups.

*DSM III*[3] lists the disorder as one of the pervasive developmental disorders. These disorders are characterized by marked abnormalities in the rate, timing, and sequence of many basic psychological functions. The six criteria for infantile autism are:

1. Onset before 30 months
2. Pervasive lack of responsiveness to other people (autism)
3. Gross deficits in language development
4. If speech present, peculiar speech patterns
5. Bizarre response to environment (eg, resistance to change, peculiar interest in or attachments to inanimate objects)
6. Absence of delusions, hallucinations, loosening of associations, and incoherence, as in schizophrenia

The relationship of the various symptoms to both chronological and mental age, however, is an important source of diagnostic unreliability. Some behaviors may be present at one age but not at another. The only symptoms which seem clearly to differentiate the autistic group are degree of deviant social withdrawal and degree of deviant communication.

These *DSM III*[3] diagnostic criteria are difficult to quantify and difficult to apply. A "pervasive lack of responsiveness to other people" is an important criterion, but by school age, many of these children have formed real, albeit deviant, relationships. *DSM III*[3] overemphasizes speech per se, since many autistic children never speak at all, and all show deficits in nonverbal communication. Many autistic children would satisfy the prodromal criteria for a diagnosis of schizophrenic disorder. Finally, some hold strange beliefs which come close to being delusional.

In attempting to delineate the common core behaviors necessary and sufficient to make the diagnosis, and to define them operationally, a recent workshop on the diagnosis of autism and related disorders concluded that deviations in social responsiveness and interaction were the core characteristic, and that impairment in both verbal and nonverbal communication was secondary.[4] The presence of repetitive behaviors and abnormalities

in play were not considered sufficient for the diagnosis, but in the presence of social and communicative impairments, their presence was considered confirmatory.

The revised version of *DSM III (DSM III-R)* will probably list only two types of pervasive developmental disorders: (1) infantile autism, which will be defined somewhat less rigidly than in *DSM III*, and (2) other pervasive developmental disorders. These will now be AXIS II diagnoses, as will mental retardation as well as the specific, more limited developmental disorders.

## Prevalence

In comparing all prevalence studies in Denmark, England, Wales, and the USA, Wing et al[5] found the estimated prevalence rates for children under 15 years of age to be four to five children per 10,000. This same figure was also obtained in a more recent Swedish study.[6] Studies utilizing diagnosed cases only provide lower estimates. More males than females have the disorder, in a ratio of about 4:1.[7] The prevalence rate in children with congenital rubella is 741 per 10,000.[8]

## Natural History

Most autistic children have cognitive impairments which persist throughout their lives.[9,10] Although the level of intellectual functioning varies, the majority function within the retarded range: about 60% have IQs below 50, and only 20% have IQs above 70. There is no consistent evidence to support the contention that these children obtain a low IQ score because they are inattentive or oppositional: The majority of children whose mental retardation was initially considered functional have been found to remain retarded in later years.

Considerable evidence suggests that language deficiency is a central feature of the syndrome. At least 28% of autistic children are mute, and speech delay is one of the most frequent and early complaints of the parents.[11] These deficits tend to persist. However, it is as yet unclear whether communication or cognitive deficits are more central.

These children show markedly different use of communication and of skills which underlie or precede language. They fail to show much social imitation, fail to wave "bye-bye," do not participate in imitative games, and are delayed in the way they use objects. They are usually unable to use symbolic gestures or to mime. If they develop speech, they may reverse the pronouns *I* and *you*, may echo or show abnormalities of speech rhythm, or may use neologisms. This differs from other types of speech disorders. Hence, it would seem that the major problem is not in language per se, but rather in communication.

The language of autistic children differs from that of aphasic children in five major ways, with the autistic group manifesting greater frequency of deviant language (echolalia, pronominal reversal, etc), more severe deficits in understanding, inappropriate or lacking spontaneous social speech, poorer skills in comprehension, and a lack of intent to communicate. Autistic speech may take the form of a direct verbatim recital of what has been heard, without the extraction of either general linguistic rules or meaning. These children can recall random strings of words as effectively as they do meaningful sentences. Finally, a central problem seems to be a specific difficulty in the use of symbols.

**Language deficiency is a central feature of the syndrome.**

The cognitive variables of language and IQ are important in that they predict the child's eventual adjustment. Meaningful speech before six years of age appears crucial,[12] and the higher the IQ, the more normal will be the child's later adaptation.[11]

The social development of these children has a number of striking characteristics. They tend not to seek attachments. They do not use eye gaze as do normal children to signal social intentions, such as looking into a parent's face when they are spoken to or want something. They tend not to go to their parents to greet them when the parents return home, or to go to them when hurt or upset. As the child grows older, these impairments seem less obvious, but these children still tend to show a lack of empathy, a failure to perceive others' feelings or responses, and a failure to make personal friendships. Probably more than anything else, it is reciprocity in social interaction that is missing in this condition. It is curious that, until recently, such social abnormalities have been the least studied of all the

features of autism, although it is from them that the syndrome, autism, was named.

Typically, the play of these children is stereotyped and nonsocial, with little variety or imagination. They rarely engage in make-believe games and have little ability to imitate. In middle childhood, many have unusual preoccupations, such as memorizing bus routes or numbers. Sometimes, this preoccupation is manifested in the ritualistic repetition of questions.

Stereotyped, repetitive movements such as hand or finger mannerisms, are often present, but are more frequent in those who are retarded.

Attentional dysfunctions generally decrease with age. If poor eye contact is a function of some deficit in visual fixation or attention rather than purposeful avoidance of eye contact, this may account for its apparent normalization with age.

Most autistic children have intact sensory equipment, but respond to sensory input in all modalities, including vestibular, in a distorted manner. These distortions include hyper- and hyporesponsivity, sometimes with both extremes manifesting in the same child.

**In most cases, the child's development has been abnormal from the beginning.**

The present outlook for autistic children is generally poor. In his review of follow-up studies, Lotter[13] reported 5% to 17% as able to develop a normal social life and to do satisfactory work as adolescents and adults. In contrast, 61% to 74% were incapable of leading independent lives.

Whether infantile autism is the earliest expression of schizophrenia, as was once believed, needs finally to be rejected or verified through systematic longitudinal studies, but considerable evidence already exists that it does not. For example, one recent follow-up study of 14 adults, most of whom were high functioning, revealed a high incidence of poverty of speech, poverty of content of speech, perseveration, and affective flattening, but revealed them to be very different from adult schizophrenics, or child schizophrenics as adults.[14]

## Etiology

The first attempts to understand autism postulated it as a disorder of affective or social functioning. Kanner, for example, called it a "disturbance of affective contact," and conceptualized the child's "pervasive aloneness" as an attempt to avoid the intolerable effect of intrusion by others. This certainly fits with clinical observations of gaze aversion, a failure or refusal to make eye contact with others, the autistic infant's lack of anticipatory posture changes when approached to be picked up, and his lack of molding to the mother's body when held.

From the late sixties, however, the popularity of this traditional view of autism as primarily a disorder of attachment and affect has declined, to be replaced by the postulation of some central cognitive deficit, whether this be in reticular arousal or hypoactivity; in relating incoming stimuli to memory; in processing symbolic or sequential information; in remaining "stimulus bound," incapable of the internal, symbolic manipulation of stored images; or in some other abnormality. However, social isolation of this severity is rare in brain-damaged children.

The popularity of psychogenic theories of autism has declined for lack of supportive evidence.[15] A recent study by Koegel, Schriebman, O'Neill, and Burke,[16] for example, showed that families of autistic children do not differ from "normal" families in terms of parental stress, family interactions, MMPI profiles (a personality inventory), or marital happiness.

While there is little good evidence to support psychogenic theories of autism, there is a growing body of literature to support biogenic ones. Twin studies show a significant genetic predisposition,[17] and it is estimated that the rate of infantile autism in siblings of autistic children is 50 times greater than in the general population. Probably it is not autism that is inherited, but rather some broader predisposition to abnormalities in social, cognitive, and language development.

Autism can also arise from a number of differing neuropathological conditions, including tuberous sclerosis, toxoplasmosis, encephalitis, lead intoxication, congenital rubella, cerebral lipoidosis, phenylkentonuria, and fragile-X syndrome, among others. Although these diseases share no obvious crucial feature, there is consistency in the conditions which are and are not associated with autism. The number of children with abnormal EEGs is higher than once suspected, and 25% or more develop seizures by

adulthood. It can be argued, it seems, that autism may represent a collection of symptoms found in a variety of handicapping conditions.

In comparison to normal children, autistics have more physical signs suggestive that something is wrong with the central nervous system. However, attempts to localize lesions have not been successful, although it does appear that there may be some association with abnormalities in hemispheric functioning, perception, or attention. Recently, neuropathological findings of six of seven brains of autistic children[18] revealed lower Purkinje cell counts in the cerebellum, a potentially important finding in view of the short-latency tract from the fastigial nucleus to the septal complex, suggesting a mechanism for limbic involvement. Early damage in this area perhaps could differentially affect development of right and left hemispheres, producing the picture which has been reported.

**There is no single proven treatment or treatment regimen for autism.**

In the *DSM III* system, age of onset is considered important in differentiating infantile autism from childhood onset pervasive developmental disorder. This appears to rest on the work of Kolvin, but depends in most cases on parental reports which may not be fully accurate. In most cases, the child's development has been abnormal from the beginning, but in about one fifth of cases, there appears to have been a brief period during which the child developed normally.

Cross-comparisons of endocrine and biochemical studies are difficult because of differences in diagnostic categories. Such factors as age, activity level, sex, treatment of intellectual level, and organic neurological or medical disease need to be considered in the evaluation of any parameter. High levels of serotonin, for example, seem more strongly correlated with low intelligence than with the diagnosis of autism.

Thus, it appears that autism is a final common symptom picture, the end-product of an underlying deficit or combination of deficits that damage cognitive and perceptual functioning and emotional responsiveness. This does not deny, however, that social and psychological factors cannot aggravate the disorder. Yet to be explained is what makes one child with brain dysfunction autistic, another retarded, and why some show no associated Axis III diagnosis.

## Differential Diagnosis

It is important to approach the question of differential diagnosis in a stepwise fashion, because of the problems of getting lost in the plethora of possibilities.

The first step requires determination of the child's current nonlanguage intellectual level. The next step is to determine his level of language development. The next step is to consider whether the child's behavior is appropriate for his chronological, mental, and language age. If not, we can consider differential diagnosis of psychiatric disorders according to patterns of social interaction, language delay, and other behaviors.

Early infantile autism usually occurs without any clear-cut period of normal development. If there has been normal development up to age 2 to 2½ years, disintegrative psychosis, elective mutism, acquired aphasia, and schizophrenia in childhood should be considered.

If the child's development has been abnormal from the beginning, an abnormal pattern of bonding secondary to parental neglect or abuse and the differential diagnosis of language delay (table) need to be considered. The differentiation may be most difficult with those children who clearly are severely retarded, because it is difficult to determine if development is deviant as well as delayed.

## Treatment

Despite the claims of the advocates of certain approaches, there is no single proven treatment or treatment regimen for autism. Some interventions, however, appear more useful than others. Indeed, the recognition that these children are not schizophrenic or emotionally withdrawn but suffer from a severe and widespread developmental disorder of constitutional origin led to rather radical changes in treatment approaches during the seventies and early eighties.

In designing an appropriate plan of treatment, five main goals need to be kept in mind:

1. Elimination of maladaptive behaviors

| Differential Diagnosis of Infantile Autism | | | | | | |
|---|---|---|---|---|---|---|
| | **Infantile Autism** | **Mental Retardation** | **Hearing Impairment** | **Environmental Deprivation** | **Developmental Language Disorder** | **Elective Mutism** |
| **Language Comprehension** | Impaired vocabulary. Grammar may be affected less than vocabulary | Impaired vocabulary and grammar | Mild to severe impairment | Impaired for age. Vocabulary especially limited | Mildly to severely affected. Vocabulary and grammar equally affected | Normal |
| **Language Expression** | Very limited, echolalia & jargon. Content often bizarre, stereotypic, or literal | Development follows normal patterns but each stage very long. Echolalia only with serious comprehension problem | Pattern of acquisition normal | Slow for age. May show jargon and echolalia | Normal | Normal, but delayed |
| **Nonverbal Functioning** | Retarded to bizarre use of toys/objects. Impaired creative play | Limited | Appropriate. Nonverbal ability normal | Normal | Normal | Normal |
| **Social/Behavioral Functioning** | Uninterested in communication. Understands but rarely uses gestures. Resistance to change | Interested in communication. Behavior grossly normal except in severe cases | Grossly normal except in severe cases | Appears shy and anxious | Normal | Normal, but child may be oppositional and stubborn |

2. Reduction of rigidity and stereotypy
3. Fostering of normal development — especially in regards to socialization, cognition, and language
4. Promotion of learning generally — especially in view of the lack of self-direction shown by these children
5. Alleviation of family distress

For each of these goals, a specific series of graded changes needs to be designed. In essence, this means the bringing about of major changes through a series of rather small steps. However, each of these goals needs to be designed specifically for each child and his family. For example, mute children lacking prelinguistic skills tend to show little improvement even with intensive treatment in language; they are more likely to profit if effort is put into the development of comprehension rather than expressive speech.

The most effective interventions are those which are started early and are continued throughout the child's life. The treatment of choice is a systematic, intrusive, educational and behavioral approach. This requires early diagnosis, early counseling of parents, well-structured and individualized special education, and involvement of the parents in the child's treatment.

Applications of behavior modification principles are effective in teaching speech, self-help, and social skills and in enhancing adaptive functioning.[19] The children who respond best usually have parents who are willing to become primary therapists. Many communities now have special educational programs designed to meet the needs of these children. These

usually focus on cognitive skills and speech as well as adaptive and social skills.

There has been increased emphasis in finding an effective biological treatment. A number of drugs have been tried, but none has been successful in eliminating the basic symptoms of autism, although some antipsychotics, eg, haloperidol and trifluoperazine, have been shown helpful in modifying associated problems such as sleep disturbances, attention problems, and stereotypic behaviors. Generally, the sedating agents seem more associated with increased withdrawal and apathy. Stimulants tend to cause greater disorganization and an increase in stereotyped behaviors. Fenfluramine, a drug which reduces serotonin levels, has been associated with behavioral improvements in children with both elevated and normal serotonin levels.[20]

With current research suggesting organic causation, attention has shifted from influence of the family on development of autism to the impact of the autistic child on his family. The major outcome studies of the last thirty years have shown that most autistic adults (60% to 70%) live dependent on relatives or institutions. Only 1% to 2% achieve a normal independent status. The parents often become worn out by the time the child reaches adolescence and seek placement. Indeed, a recent book by Schopler and Meisbov, *The Effects of Autism on the Family,*[21] deals with this topic. For these families, local chapters of the National Society for Autistic Children can provide important sources of social support and practical advice.

## Summary

Systematic follow-up studies have revealed that autism is a chronic disorder characterized by modest symptomatic improvement but with residual social and, frequently, cognitive and linguistic impairments. Disagreements over syndrome definition and diagnosis have impeded research efforts. *DSM III* broke new ground with its emphasis on developmental disorders and its multiaxial diagnostic framework, but the system did fall short of its goals.

Although infantile autism was first described as a disorder of affective contact, it has, in the past twenty years, been regarded primarily as a cognitive disorder, with the socially deviant behavior considered secondary. Now, however, there seems to be more generally accepted agreement that social relatedness and communication are deviant, not just delayed, and that at least the defects in social relatedness are primary. Only with more clearly operationally defined diagnostic criteria and clearer delineation of more homogeneous subtypes within the syndrome will it be possible to address adequately the more interesting questions of pathogenesis and treatment.

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# Occurrence of Bacteria from Blood, Wounds, Urine, and Sputum of Patients at a Veterans Administration Medical Center (1975-85)

D. J. FLOURNOY, PHD

**Routine (not including acid-fast bacilli) bacterial culture results were compiled annually from 1975 through 1985 at a Veterans Administration Center. The six most common organisms from blood, wound, urine, and sputum specimens were determined. Overall, the most common isolates were *Staphylococcus aureus* (blood and wounds), *Escherichia coli* (urine), and *Haemophilus influenzae* (sputum). Some isolates like *S aureus* (wounds) and *E coli* (urine) were always the most common, while others like *S aureus* (blood) and *H influenzae* (sputum) varied in their occurrence over the years.**

Bacterial isolates at each hospital are somewhat unique and reflect the patient population and peculiarities of the institution. Periodic quantitation of isolates is one way of evaluating the importance of organisms. This information can be useful to many individuals including medical technologists, physicians, administrators, and others within and even outside of the institution. Certain organisms may be considered important based on their occurrence, pathogenicity, or costs for related diagnostic tests.

Information gained from analyzing the occurrence of bacterial isolates can be helpful as a teaching subject, establishing priorities and detecting long- or short-term (eg, outbreaks) trends. If an organism is commonly encountered, increased efforts relating to efficiency of its identification, treatment, and/or elimination might decrease patient morbidity, mortality, and related costs.

Microbiology is still a somewhat expensive, labor intensive discipline, although some automation is available. When clinical laboratory funds are limited, it is imperative that laboratory's departments operate efficiently in order to produce the most relevant diagnostic test results at the lowest price. Finally, individual hospital isolate occurrence should be historically documented in order to allow analysis at a later date, if desired.

This report notes organism prevalence in a single institution, primarily serving adult males, during a period of eleven years.

## Materials and Methods

The Veterans Administration Medical Center (VAMC) in Oklahoma City is a 434-bed general medical and surgical hospital primarily serving adult males in Oklahoma and north central Texas. During the study period, there were from three to five full-time medical technologists, a media maker, and a director working in microbiology. Also, the Microbiology Section was approved as an Extent IV Laboratory in Bacteriology by the College of American Pathologists.

(continued on p 305)

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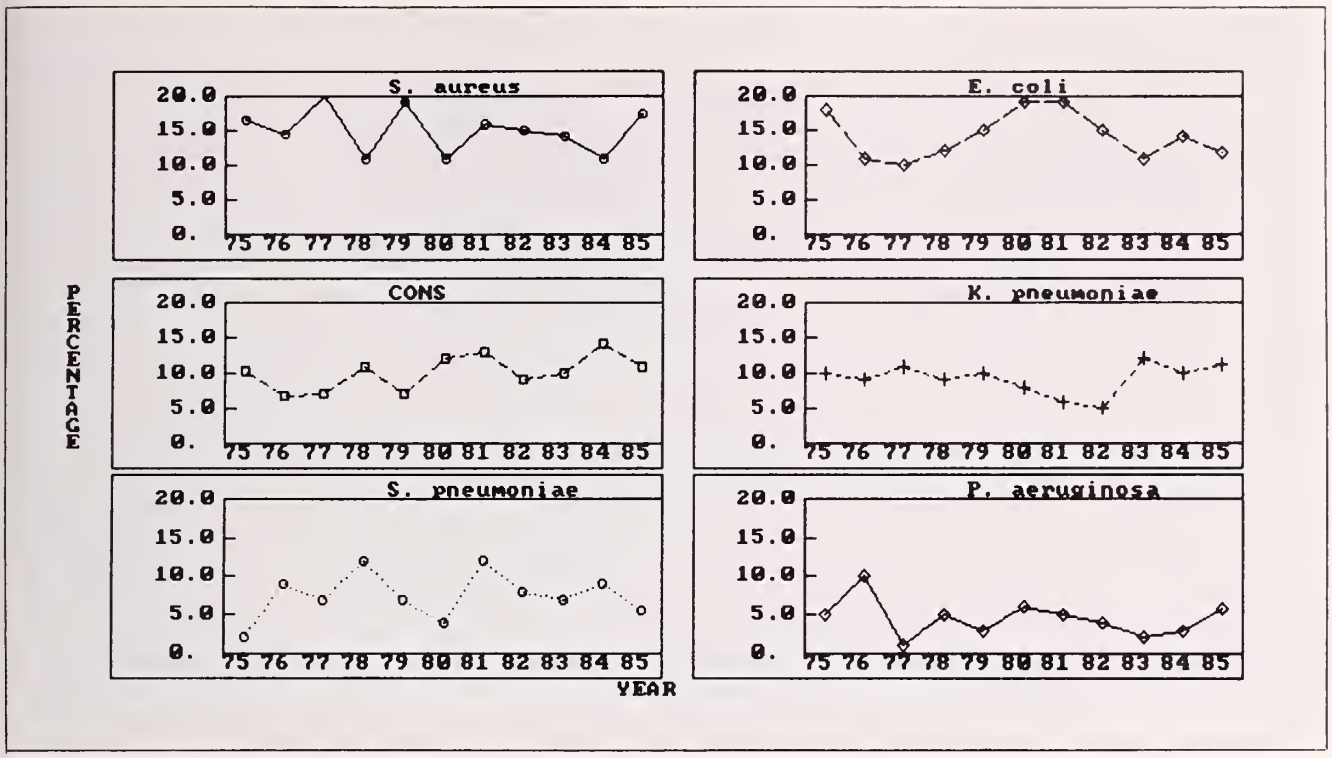


**Fig 1.** Blood culture isolates. Order of occurrence was: most common (top left), 2nd (top right), 3rd (middle left), 4th (middle right), 5th (lower left), and 6th (lower right).

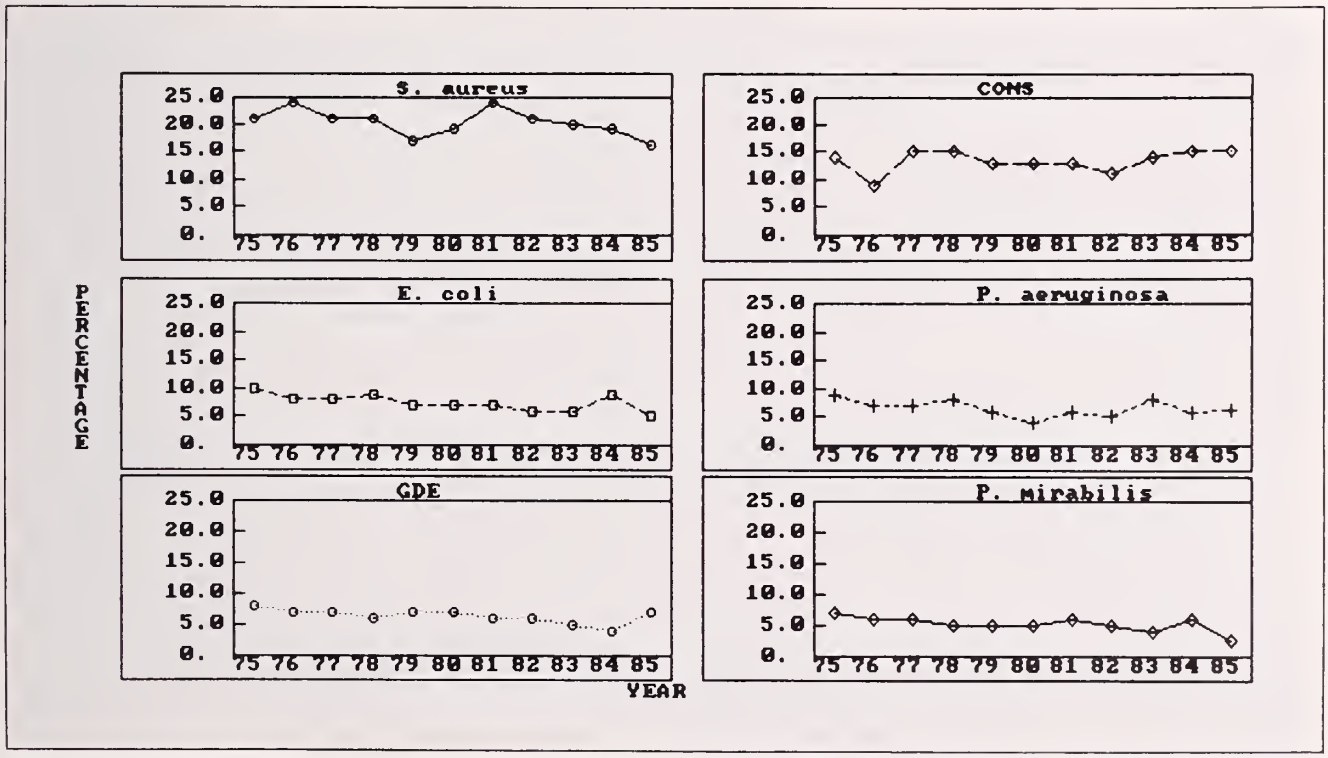


**Fig 2.** Wound culture isolates. Order of occurrence was: most common (top left), 2nd (top right), 3rd (middle left), 4th (middle right), 5th (lower left), and 6th (lower right).

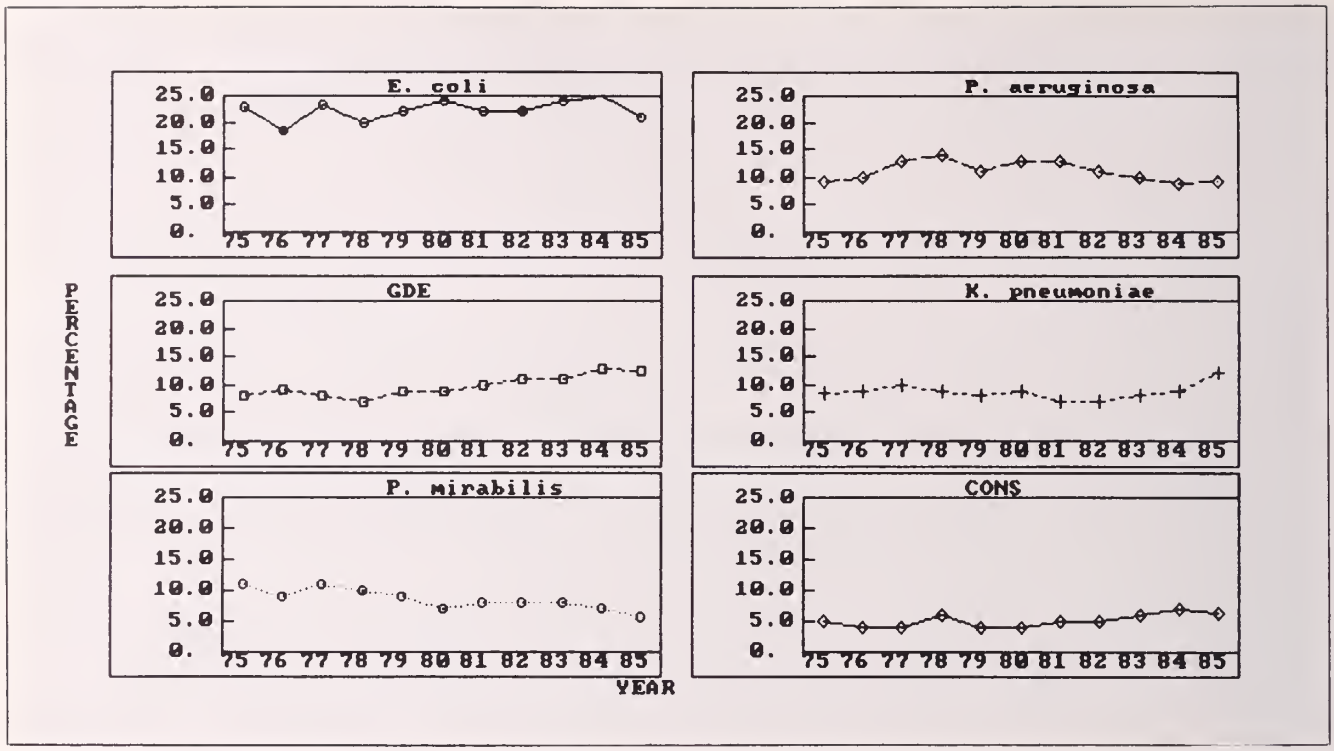


**Fig 3.** Urine culture isolates. Order of occurrence was: most common (top left), 2nd (top right), 3rd (middle left), 4th (middle right), 5th (lower left), and 6th (lower right).

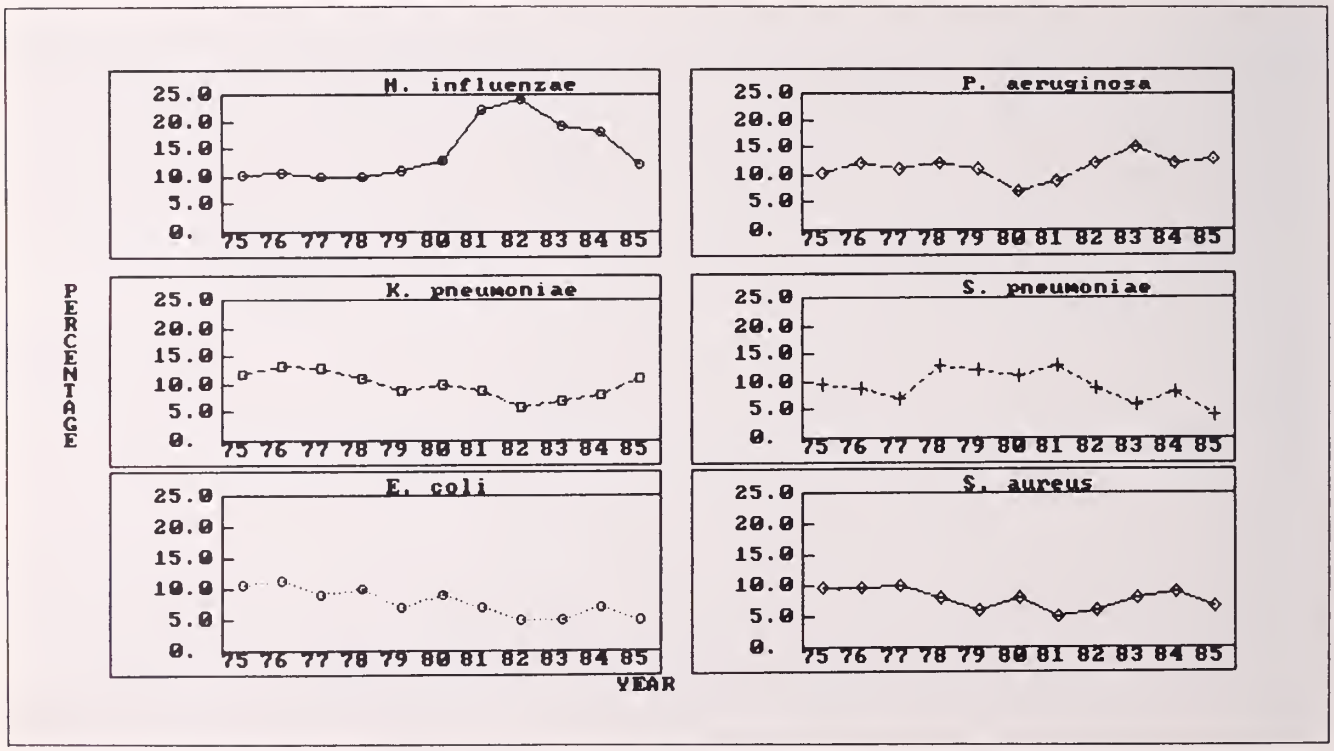


**Fig 4.** Sputum culture isolates. Order of occurrence was: most common (top left), 2nd (top right), 3rd (middle left), 4th (middle right), 5th (lower left), and 6th (lower right).

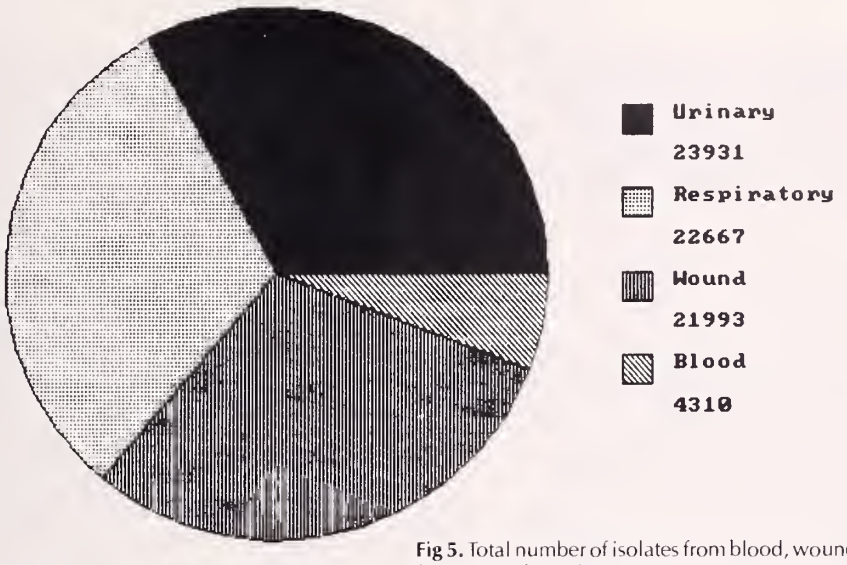


**Fig 5.** Total number of isolates from blood, wounds, urine, and sputum from 1975 through 1985.

Bacteria were isolated and identified by standard accepted procedures.[1-3] Data were recorded daily in microbiology logbooks, and isolate occurrence was compiled annually from 1975 through 1985. The data represented a number of isolates; therefore, in some cases, an organism may have been isolated on different days and counted separately each time.

In general, total isolates are represented for blood, urinary, and wound cultures, whereas only significant isolates are noted for sputum (respiratory) cultures. Urinary tract isolates, from clean-catch, midstream specimens, were counted if in a concentration of $1 \times 10^4$ colony forming units or more as determined using the 0.001 ml calibrated loop technique. Not all sputum, urine, wound and blood isolates that were judged, by microbiology section personnel, to be normal flora or contaminants were counted. In some cases where opportunistic organisms (eg, coagulase negative staphylococci, CONS) were encountered, the figures represent both pathogens and contaminants. The Fisher Diagnostics Blood Culture Bottle-SPS (Orangeburg, NY), orignally manufactured by Lederle Diagnostics, Inc, was used throughout the study. The term *wound* included all miscellaneous patient cultures like fluids and body sites.

## Results

The occurrence of the six most common isolates for each specimen type are presented as follows: blood (Fig 1), wounds (Fig 2), urine (Fig 3), and sputum (Fig 4). The percentage of most organisms remained somewhat constant for wounds and urines. Sputa exhibited a drastic increase in *H influenzae* isolates beginning in 1981. The occurrence of blood culture isolates was also somewhat erratic, perhaps because there were fewer total isolates than with the other specimens; therefore, changes produced larger percentage shifts.

Figure 5 compares the total number of isolates for the different types of specimens. Urines, sputum, and wounds yielded similar numbers, whereas blood had fewer.

**The great majority of disseminated infections or bacteremias are hospital acquired.**

For 1985, the number of cultures was: urines (6097), sputa (3112), wounds (2698), and bloods (5399). The percentage of significant positive cultures was: urines (33), sputa (59), wounds (69), and bloods (10). When exudates were separated from wounds, there were 1065 cultures with 14% positive.

The total number of routine bacterial cultures

was compared with the number of hospital admissions (including urine dialysis) by year (Fig 6). These two parameters appear to be somewhat directly related. In 1982, the laboratory service moved into a new laboratory, and admissions were limited due to construction.

## Discussion

It is important that some long-term studies of organism prevalence in individual institutions are documented. Large studies which combine data from several/many institutions are also important, but they inherently possess more variables due to differences among hospitals. These variables can obscure clear-cut interpretations.

The nosocomial infection surveillance survey from the Centers for Disease Control (Atlanta, Ga) is useful as a comparative document, although it only lists nosocomial pathogens.[4] The survey noted that *E coli, S aureus,* enterococci, and *P aeruginosa* were the most frequently reported pathogens. By site, the most frequent nosocomial pathogens were: urinary tract (*E coli*), surgical wounds (*S aureus*), lower respiratory tract (*P aeruginosa*), and primary bacteremia (coagulase negative staphylococci). Since these organisms were also common in the VAMC, a comparison of results suggests that nosocomial pathogens probably represent a significant proportion of our total isolates. However, it is interesting to note that most lower respiratory and genitourinary infections are community acquired, whereas the great majority of disseminated infections or bacteremias are hospital acquired.[5]

The VAMC in Denver reported that its six most common isolates in descending order were: *S epidermidis, E coli, S aureus, S pneumoniae, K pneumoniae,* and *P aeruginosa* for 1975 through 1977. These were also our most common blood culture isolates, but in a different order.[6] In our hospital, coagulase negative staphylococci were the third most prevalent group of organisms. This suggests that there are similarities in isolates among hospitals serving adult males, although some differences occur.

Although this report does not include antimicrobial susceptibility data, a recent publication has noted trends in susceptibilities from 1975 through 1982 among our isolates.[7] In general, most gram negative bacilli exhibited increased susceptibilities, whereas *S aureus* and Group D Enterococci showed some decreased susceptibilities. Also, another report covered bacteremia at this hospital from 1961 through 1981.[8]

Results from this study and many others suggest that increased attention should be paid to the rapid

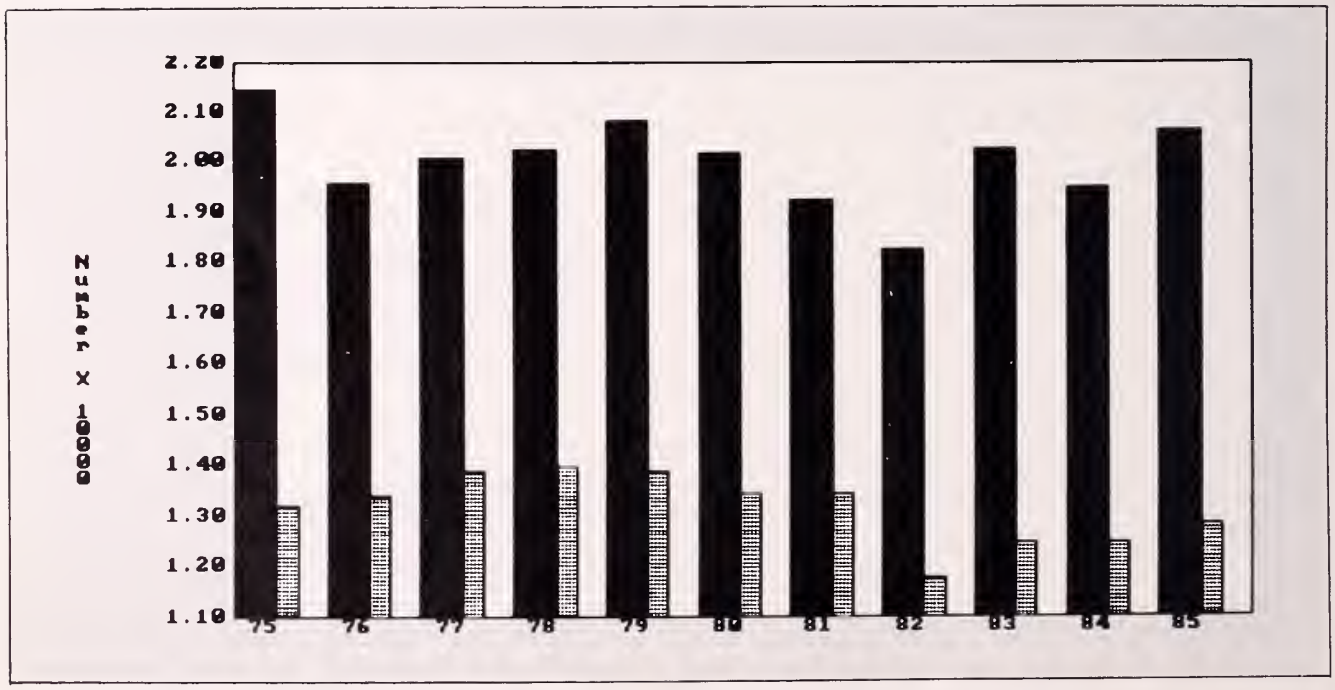


**Fig 6.** Comparison of routine bacterial cultures (dotted) and patients admitted (solid) by year from 1975 through 1985.

identification of common organisms like *S aureus, E coli,* and *H influenzae* (at our hospital). Antimicrobial susceptibility results should be provided as quickly as possible in life-threatening infections (ie, bacteremia), and the physician should be given a choice of appropriate drugs (at least three) to choose from. If pathogens are resistant to routine drugs, additional new drugs should be tested. The use of isolate occurrence data can allow laboratories to place appropriate emphasis on primary pathogens, thus decreasing patient morbidity/mortality and related costs.

*Acknowledgments:* Sandy and Jean Rutherford deserve thanks for their help in data collection.

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## Coming in June . . .

Manuscripts being considered for publication in June include a paper on love and attachment and a lengthy report on outpatient geriatric assessment.

# Our Kinship with the Child-Abusing Parent (Debbie K: A Child Is Bruised)

JOHN E. POARCH, MD

**It is very difficult to complete medical school if one is not relatively compulsive. This paper is directed toward those successful physicians who are very self-critical and demanding and who find it quite difficult to sustain a reasonable degree of gratification and self-approval at work and at home in spite of great effort.**

The above title is intended to leave the reader with the impression that Debbie is a child, although she is not. This case report attempts to communicate some understanding of the child-abusing parent, in order that the reader can empathize with the parent instead of the child who is the victim. It is often very hard for us to feel anything but rage toward the abuser of children.

The report is just as it was sent to the attorney, with changes for anonymity, of course. It will help to know that I had previous contacts with the childcare worker and spoke with both attorneys and the presiding judge by conference call during the initial hearing. Everyone was very cooperative. The judge volunteered to delay adjudicating Debbie at that time.

A brief discussion of the kinship between the conscience of the compulsive and that of the abusive parent follows the report.

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## Psychiatric Report: Presenting Complaint

Debbie came to see me in December 1982. She was a slender woman who was dressed in jeans and a heavy, long, dark, coat-type sweater, with an Aunt Jemima type bandana covering her hair, and she wore little or no make-up. She was neither appealing nor attractive (although it was apparent she might be) and struck me as someone who could have spent time on the back ward of a state hospital. She was so extremely removed (detached) from her emotions that she was in grave doubt about her sanity.

She had previously been to see a marriage counselor. At that time her husband was in Saudi Arabia as an airplane pilot for an American company doing business there. He was threatening to divorce her if she did not return with the children to Saudi. She was living with her parents and very ambivalent about going back to Arabia. If she returned to her husband, she wanted to do so out of desire rather than because of his threats. There was such a feeling of urgency that I invited her to come twice per week (at the time my expectation was that within six to ten weeks she would overcome her ambivalence and rejoin her husband).

## Relevant History and Psychiatric Examination

Debbie is 35 years old and the second oldest of nine children. She is the mother of three children all under age five. There is a history of Debbie being

spanked and of her spanking her younger siblings when she was taking care of them, but no history of her being physically abused nor of her abusing younger siblings. Although the extent is unclear, there is a history of sexual over-stimulation and there was some degree of incestuous activity with a brother.

Debbie feels very alone and unsupported in bearing responsibility of the children. She is obviously of above-average intellect, but she often takes an extraordinary amount of time between words and phrases, as though sorting and organizing thoughts is a great burden.

Debbie's most prominent primary defense is detachment. At those moments when she most needs acceptance and support from within, ie, when she experiences intense emotion, she, in a sense, abandons herself (as a mother might turn off her feelings when her small child makes her very nervous) through detachment. *At these times she may seem well put together but indifferent and uncaring to the observer.* The detachment is never more pronounced than when she is experiencing extreme anger or rage, as on one occasion she became so isolated and detached as to almost faint.

The latter event happened in combination with paranoia, and in all my experience I had not seen this phenomenon before. It occurred in the session after she was first arrested. She had projected her inflexible conscience (internalized parent) onto me (the parent who abused children) and was determined to destroy it. (She had in mind a plan to ruin my reputation and saw me as responsible for not preventing the abuse.) She became like a wax dummy for about twenty to thirty seconds.

When she is angry, she experiences herself as loathsome, disgusting, unworthy, and undeserving of interest, concern, or care. Hence the abandonment of self.

From the foregoing, it is not surprising that at times of intense emotion, with the accompanying alienation, Debbie's reality testing breaks down and she may converse inappropriately, such as discussing incestuous wishes with her father and siblings. She may also then become a victim (as opposed to the moderator) of her sexual and aggressive impulses and perform bizarre or inappropriate acts.

Debbie's demands and expectations of herself are so great as to make it impossible for her to consistently have a reasonable degree of approval from her conscience. (She brought in some clothing she had made for her children and the inside seams were finished out in superior fashion.) She experiences her conscience as an inflexible, ungiving "piece of steel." Since she relates to other people the same way, this also makes it relatively impossible for them to live up to her demands and expectations — including her children and her therapist. More specifically it also means that when she most needs the support of other people, she evokes alienation and even wrath from them.

At those times of breakdown of her reality testing, she may experience one of her children as a sibling of whom she was jealous or an unloving, ungiving parent. For example, when talking of the spanking of her daughter, she made an unconscious slip and referred to her by one of her sisters' names.

It is possible for Debbie to resort to projection, but I have only seen her relatively paranoid on one occasion. To her good fortune, she did have some awareness that she was irrationally suspicious. This occurred the first visit after the bruising of her daughter's buttocks.

## Course of Therapy

Over the next few months, with empathy and understanding, Debbie came out of her isolation and detachment at such a rapid pace as to astound me. It was like watching a psychotic or extremely depressed person come back to rejoin the living. It was delightful to discover that she was attractive, coquettish, and appealing. To no one's surprise, with such dramatic and intense involvement and change (we were working in a very positive relationship), she began to express feelings of love and desire which I interpreted. Such feelings are not unusual and do not correlate with the age or physical attractiveness of the therapist, and such expressions toward a therapist are not uncommon in the course of psychotherapy. (There were statements about this referred to in the legal charges.)

During the winter and spring there were phone conversations between Debbie and her husband which were described as coercive, with each one obviously responding to the other with resistance and antagonism. My impression was that both were involved in a competitive power struggle but each was afraid of the pain of loss and separation, so that I was very surprised when she reported that Mr K had filed for divorce.

For many weeks Debbie could not really believe that a divorce was going to become a reality. She seemed to assume that reconciliation would eventually occur. It later became apparent that she was just

terrified at the prospect of facing the reality of divorce.

It should be noted that throughout our contacts there was much discussion of the children, as Debbie has a very obvious desire to be an exceptional parent; but at the same time, she expressed more than unusual doubt and uncertainty about child rearing and discipline.

As we moved through the summer and into the fall of 1983, Debbie still only partially accepted the reality of the divorce. On the occasion that she first went to a neighboring state to meet with her lawyer (because her husband had filed in that state she was required to obtain a divorce counsel there), she came home in unusual distress as this was the first time she was able to face the reality that a divorce was indeed going to take place. It was on Friday of that week that the bruising of her daughter took place, and she called her sister to alarm everyone that she was too overwhelmed to be trusted with the care of her children. It is to her credit that she screamed for help and unconsciously insisted upon others taking over when she could no longer trust herself.

## Summary and Recommendations

Please note the enclosed list of references reviewed prior to preparing this report. This task was by no means taken lightly.

Bear in mind that the best predictor is history and that if an adult is obviously out of control, it is very easy for the person to kill a small child rather than bruise it. Some degree of inner control must exist to stop and call the alarm rather than hide, as do many abusive parents.

It is very important to note that there is no history or evidence of physical abuse of the children prior to the fall of 1983, when Debbie was trying to accept the reality of divorce. Thus, only when she was facing the most severe emotional pain possible (separation or loss) did she express being overwhelmed by bruising her children. It is also important to note that the youngest was spared.

I have never felt and still do not feel that Debbie's children's lives were at critical (burns or broken bones) risk when she was alone with them. Her calling others and unconsciously insisting on police involvement is evidence which I find reassuring under the circumstances. Also, the fact that she was not physically abused, that she did not abuse her younger siblings, and that the child abuse did not occur until she was under extreme stress of single parenting and divorce is prognostically favorable. Taking care of three children under five is a tremendous job even with the presence and support of a husband and father.

I do not feel, given her background and personality make-up, that it is *currently* in her best interest and equally the children's best interest, for her to bear full-time responsibility for their care. It is to her credit that she was doing everything she possibly knew to do, including seeking expensive psychiatric help, with the sincere desire to do the very best by her children and herself that she could. It turns out it was not enough.

**The parent's attitude toward the child is the same as the conscience's attitude toward self.**

Debbie obviously has many strengths and I would hope that with lessened stress and responsibility, plus time to adjust to the divorce, there could be serious consideration given to joint custody and later full custody. I think it is important, especially now, for her to continue in therapy and I have no doubt she will. With time to adjust and the children being older (by even one to three years), their care would not be so stressful.

If children are removed from a parent because society (as adjudicated through public court) declares the parent unfit, then as the children grow older and able to understand, they begin being afraid of becoming an unfit person. The energy spent to prevent this is wasted and tends to bring about exactly what they are trying to avoid, ie, becoming an unfit parent. The consequences can influence several future generations and thus many people. Also, it is not easy, by any means, for the best of mental health professionals to try to repair the severe wound caused to the parent declared unfit.

In spite of the extreme emotional pain, Debbie is currently planning to voluntarily give custody to Mr K with the hope of gaining liberal visitation privileges and consideration of joint custody or a return of full custody at a future date.

The charges of child abuse against Debbie (with the exception of her second arrest), and the system, including Nancy Lynott with DHS, Robin Lewis at the North Care Center, Mr Horning, and Judge Manning, have performed a great service for Debbie and her children. Debbie now is no longer psychologically overwhelmed but at the same time her suffering could hardly be greater than it is. Thus, from a psychiatric standpoint, the legal charges no longer serve any useful purpose.

## Discussion

It is perhaps through understanding the interaction between the child-abusing parent and the abused child that we can come to really understand that the parent's attitude toward the child is the same as the conscience's attitude toward self and that the child's attitude toward the parent is also the attitude the person has toward their own conscience.

There is a kinship between the successful business executive or professional with a very compulsive personality and the child-abusing parent. Both have a very critical and demanding conscience, a conscience with expectations and requirements that are very difficult, if not impossible, to meet.

The compulsive personality has a conscience (internalized parent) which will give a degree of acceptance, approval, and support; but because the same degree of distance between the conscience and the self must be maintained, the person cannot feel very well for very long before the demands and expectations are increased. Thus, the degree of fulfillment or pleasure remains limited. This is why the more successful one becomes, the more successful one must become.

Frequently these people tend to feel unappreciated since they relate to others the same way they relate to their conscience. They complain about their wife, husband, boss, company, or their children not really appreciating how hard they try, and getting furious with them from time to time.

Some find it extremely difficult to boss or discipline subordinates or children. They feel so constricted and burdened by the demands and expectations of their own conscience that they feel like a monster when they are in a position which requires them to administer consequences. It leaves them feeling miserable.

The child-abusive parent's conscience gives *no* acceptance, approval, or support, no matter how well the person performs, and eventually there ensues a rage with a desire to destroy the internalized parent. The children are the most reactive people around and those reactions are looked at as definitions of self; thus, when they are negative and do not give support and approval to the parent, the wish to destroy is directed toward the child.

If we convey to our patients that their problems are their parents' fault, in a blame sense, we are wasting their time and ours. If we communicate that their problems are related to their parents' problems in a phenomenological sense, even though they may or may not have different manifestations, such as the pure and perfect mother and the alcoholic son, or the judge and his delinquent son, then we have done something perhaps worthwhile.

We must also help them understand that the problems of their parents will become their progeny's problems, although they may have different manifestations, unless they are able to face the terror that prevents change. The terror I refer to is the terror related to the child within the patient feeling like if he does change, he will be abandoned by the internalized parent.

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News from
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# Declines in gonorrhea and syphilis: Fear of AIDS affecting behavior?

Public concern with the transmission of Human Immunodeficiency Virus (HIV) increased substantially during 1986 in Oklahoma. The increased risk for individuals with multiple sexual partners, homosexual or heterosexual, to exposure to HIV should be well understood by the sexually active segment of the population. Has this information and the fear of an untreatable fatal disease changed the health behavior of the high risk groups or the general public? Changes in the numbers of reported gonorrhea and syphilis cases may be indicators of behavior modification.

Declines in reported gonorrhea and early syphilis morbidity occurred during calendar year 1986. While the overall declines are encouraging, closer scrutiny of where changes occurred may provide information for future public health planning.

Decreases in primary and secondary syphilis and gonorrhea occurred for both sexes and in every age group, with few exceptions. Increases were reported for both sexes in the age group under 20 years of age and for females greater than 29 years of age. The increases in the under-20 age group occur at a time when the proportion of the population in this age range is declining. Of particular concern for public health planning is the need for education of this group.

Specifically reviewing gonorrhea morbidity by county reveals some interesting data. In Oklahoma, 30 of the 77 counties contribute just over half of the state's total morbidity. Ten of these counties reported decreases in morbidity, while 20 experienced increases. Worth noting are decreases in morbidity reported by Muskogee County (-7%), Oklahoma County (-3%), Pittsburg County (-11%), and Tulsa County (-14%).

More effective education of high risk individuals appears necessary. Early syphilis cases in homosexual males is not declining as would be expected due to concern over HIV infection. The number of males with syphilis who reported homosexual activity increased 22% from 1984 through 1986. Homosexual males accounted for 32% of the total early syphilis cases reported in 1984; that total grew to 39% in 1986.

| DISEASE | February 1987 | TOTAL TO DATE | | |
|---|---|---|---|---|
| | | This Year | Last Year | 5 Yr. Avg. |
| AMEBIASIS | 0 | 2 | 1 | 1 |
| CAMPYLOBACTER INFECTIONS | 14 | 28 | 26 | — |
| ENCEPHALITIS, INFECTIOUS | 2 | 3 | 1 | 3 |
| GIARDIA INFECTIONS | 21 | 35 | 29 | — |
| GONORRHEA (Use ODH Form 228) | 814 | 1679 | 2131 | 2161 |
| HAEMOPHILUS INFLUENZAE INVASIVE DISEASE | 14 | 22 | 36 | — |
| HEPATITIS A | 30 | 55 | 45 | 66 |
| HEPATITIS B | 22 | 41 | 18 | 25 |
| HEPATITIS, NON-A NON-B | 5 | 9 | 4 | — |
| HEPATITIS UNSPECIFIED | 9 | 13 | 11 | 24 |
| MEASLES (RUBEOLA) | 0 | 0 | 0 | 0 |
| MENINGITIS, ASEPTIC | 4 | 8 | 7 | 8 |
| MENINGITIS, BACTERIAL (non-meningococcal, non H. Influenzae) | 7 | 11 | 11 | 10 |
| MENINGOCOCCAL INFECTIONS | 4 | 8 | 4 | 7 |
| PERTUSSIS | 11 | 13 | 14 | 7 |
| RABIES (Animal) | 1 | 3 | 8 | 14 |
| ROCKY MOUNTAIN SPOTTED FEVER | 0 | 0 | 0 | 0 |
| RUBELLA | 0 | 0 | 0 | 0 |
| SALMONELLA INFECTIONS | 14 | 29 | 38 | 45 |
| SHIGELLA INFECTIONS | 6 | 29 | 22 | 30 |
| SYPHILIS (Use ODH Form 228) | 11 | 25 | 33 | 32 |
| TETANUS | 0 | 0 | 0 | 0 |
| TUBERCULOSIS | 16 | 25 | 29 | 38 |
| TULAREMIA | 0 | 3 | 0 | 0 |
| TYPHOID FEVER | 1 | 1 | 0 | 0 |

| Diseases of Low Frequency | Total to Date This Year |
|---|---|
| ACQUIRED IMMUNE DEFICIENCY SYNDROME | 9 |
| BRUCELLOSIS | 0 |
| LEGIONNAIRES DISEASE | 3 |
| MALARIA | 1 |
| REYE SYNDROME | 1 |
| TOXIC SHOCK SYNDROME | 6 |

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# M. Joe Crosthwait, MD, becomes association's 82nd president

Midwest City physician M. Joe Crosthwait, MD, became the 82nd president of the Oklahoma State Medical Association on May 2, 1987. He succeeds Norman L. Dunitz, MD, of Tulsa. Dr Crosthwait was installed at a formal inaugural dinner-dance held in the ballroom of the Sheraton Century Center during the OSMA's Annual Meeting in Oklahoma City.

A family practitioner, Dr Crosthwait has served as chairman of the OSMA Council on Professional and Public Relations for the past 14 years. He has also been a member of the Oklahoma delegation to the American Medical Association since 1972, acting as delegation chairman since 1980. He was chairman of the OSMA Board of Trustees from 1972 to 1974.

Born in Oklahoma City in 1923, Dr Crosthwait was graduated from the University of Oklahoma School of Medicine in 1955; he served his internship at University Hospital. In 1967 he was voted president of the Oklahoma City Clinical Society, and two years later, in 1969, he was elected to the board of directors of the Oklahoma County Medical Society, a position he held for three years.

Dr Crosthwait became chief of staff at Midwest City Hospital in 1970. The following year he was named president of the Mid-Del Clinical Society and in 1977 was appointed instructor of medicine at the University of Oklahoma.

Dr Crosthwait and his wife, Judy, have four children, M. Joe, Jr., an attorney, Jane Hendren, a pharmacist, and sons Jason and Todd, of the home.

*"Now it's even better"*

# JOURNAL wins again in Sandoz medical journal competition

The JOURNAL of the Oklahoma State Medical Association has won first place in the 1987 medical journalism competition conducted by Sandoz Pharmaceuticals. This marks the second time the JOURNAL has earned top honors in the competition, having won also in 1978. In 1983 the JOURNAL received an honorable mention.

A Sandoz representative presented the $500 cash prize and certificate to Managing Editor Susan Harrison on May 1, during the OSMA's Annual Meeting in Oklahoma City. Mark R. Johnson, MD, the JOURNAL's editor-in-chief for 19 years, could not be present.

Judges praised the JOURNAL's new format, introduced by Harrison in 1986. Paul Fisher, professor at the University of Missouri School of Journalism, called the JOURNAL a "very, very well done publication" and an "altogether first-rate job." A Sandoz press release said the JOURNAL "has been excellent for many years; now it's even better."

This is the twelfth year for the Sandoz competition, in which awards are given for outstanding design and editorial qualities. Twenty-four prizes were awarded this year to publications in four categories — state medical journals, county and city medical publications, state pharmaceutical publications, and journals and other nonemployee publications produced by hospitals and medical groups.

Runners-up in the state medical journal category this year were *Michigan Medicine* and *Virginia Medical*, $250 special awards; and *Iowa Medicine*, *Missouri Medicine*, and *The Ohio State Medical Journal*, honorable mentions.

**SB 134 stillborn** Concerned OSMA members and staff watch on March 9 as proponents of the tort reform bill fight to keep it alive in the Senate Rules Committee. In the gallery, l to r, are Kenneth W. Whittington, MD; James B. Pitts, MD; Marvin K. Margo, MD; and M. Joe Crosthwait, MD. Seated on the Senate floor are OSMA Executive Director David Bickham and, behind him, OSMA Director of State Legislation Otie Ann Carr. Standing next to Mr Bickham is Robert W. Baker III, OSMA associate director. SB 134 died in committee a few days later, the result of a tie vote. Senate President Pro Tem Rodger Randle, who had promised to support the bill, was not present to cast his tie-breaking vote.

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## Free program explains functions of State Board of Medical Examiners

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These and many other questions are being answered in a program presented free of charge by the Oklahoma State Board of Medical Examiners. The program will be arranged upon request for hospital staffs, medical societies, specialty groups, and civic organizations.

Part of an ongoing educational effort, the program informs physicians about the board's activities and addresses the growing problems of drug abuse, poor prescription writing habits, sloppy record keeping, and impairment in the medical community.

Inquiries about the program should be directed to the educational program coordinator at the board, PO Box 18256, Oklahoma City, OK 73154-0256, (405) 848-6841. In addition, visitors are always welcome at the board's office, 5104 North Francis, Suite C, Oklahoma City.

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*Late renewals pay penalty*

## June 10 is deadline for renewal of Oklahoma state medical licenses

Under the provisions of the Oklahoma Medical Practice Act, all Oklahoma medical licenses must be renewed on or before June 10 each year. After that date, a $10 penalty for late payment is added to the renewal fee.

On July 1, all unrenewed licenses are placed on inactive status. Reactivation of these licenses requires payment of a $150 reactivation fee in addition to the renewal fee of $100.

The most common reason for failure to renew is the physician's neglecting to notify the Board of Medical Examiners of a change of address. Renewal notices are mailed to the last address of record.

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# Cheeseburger and fries off limits to hypertensive Tulsa teens

Authorities now are saying that what physicians once thought of as normal adolescent blood pressure may be neither normal nor desirable in young people.

This news has prompted three Tulsa area organizations, Adolescent Medicine Associates (AMA), Hillcrest Medical Center, and the University of Oklahoma Tulsa Medical College (TMC) to join forces in screening area high school students for high normal blood pressure and hypertension.

On March 11, blood pressures of all students at Edison High School were checked as part of the joint screening effort. Over 50 teams of health professionals visited Edison homerooms to check 1,300 students during a one-hour period. The Edison PTA assisted the physicians and TMC faculty, medical residents, and students.

Parents were notified by letter five days prior to the screening. On the day of the screening, each student was given a card reporting his or her blood pressure, along with educational material explaining high blood pressure. Results were also mailed to parents.

Before the screening, Edward J. Tomsovic, MD, TMC dean, said, "We are hopeful that we can detect high or undesirable blood pressures in these young people. If results are questionably high, we will advise students to see their family's physician for a check-up. In most cases, simply modifying the diet and increased exercise is all that it takes to control high normal blood pressure in teens. Medication is usually not needed."

Tomsovic also noted that taking 1,300 blood pressures in one hour is quite a feat, adding that only the joint efforts of Hillcrest, TMC, AMA, and Edison PTA volunteers made the event possible.

Adolescent Medicine Associates has received a grant to study blood pressure among young people, and the results of the Tulsa screening will be part of a nationwide study.

Edison High School was chosen for the screening in connection with the Tulsa Public Schools Adopt-a-School program, in which Hillcrest Medical Center has adopted Edison. Hillcrest works with Edison on various community projects.


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# Increased hospitalization for elderly blamed on PPS, DRGs

Medicare's prospective payment system (PPS), based on diagnosis-related groups (DRGs) may be leading to increased hospitalization of the nation's frail elderly, as well as shorter length of hospital stays, suggests a study of Wisconsin's nursing homes, published in *Journal of the American Medical Association*. The study also found an increase in the proportion of deaths occurring in nursing homes and a decrease in the percent of hospital deaths from 1982 to 1985, suggesting less aggressive treatment of the terminally ill.

Mark A. Sager, MD, and colleagues, of the University of Wisconsin, Madison, reviewed hospitalization and mortality data for Wisconsin's Medicaid and generally elderly populations for one year after implementation of the DRG system. The program was started in October 1983 as a means of curbing Medicare's escalating costs.

"During the 12 months following implementation of the PPS, Wisconsin's institutionalized elderly Medicaid population experienced a 72% increase in the rate of hospitalization and a 26% decline in hospital length of stay," the researchers say. "Two explanations for the increased hospitalization include physician manipulation of the PPS and increased rehospitalization of nursing home residents who may have been discharged prematurely from hospitals."

The study indicated modest decreases in hospital admissions *and* length of stay for the noninstitutionalized elderly population. "The fact that the changes in hospitalization were seen only in the institutionalized . . . study group raises the possibility that the frail elderly may represent a unique segment of the Medicare population who are vulnerable to the changes in health care delivery encouraged by Medicare's PPS," the researchers say.

In an accompanying editorial, Carolyne Davis, PhD, former administrator of the Health Care Financing Administration in Washington, agrees that studying Medicaid records as a subset of Medicare beneficiaries helps to identify a special group of the frailest elderly, and that problems with the PPS system might be seen earlier within this group. Davis raises some objections, however, to the authors' interpretation of their data.

"It is unfortunate that Wisconsin's Medicaid records were not kept in such a fashion as to allow comparison with data from 1985 and beyond. One is left not knowing if the results are part of a transition issue, an aberration in data collection for a single year, an actual impact of system changes, or the result of flawed methodology," Davis says. She notes further that since there was an overall decrease in all beneficiary admissions (both Medicaid and Medicare), the reported 72% rate increase for institutionalized elderly is suspect.

Concerning the shift in the proportion of elderly dying in nursing homes rather than in hospitals, Davis suggests, "Although it is true that the impact of a new incentive payment system may have heightened physicians' awareness of cost-benefit issues with regards to the treatment of terminal illness, it is also true that important sociological trends were at work during this time." She adds that it is unlikely that the 1983 shift in location of death represents Wisconsin physicians' response to the advent of a new payment system, since it did not take effect until October.


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## Automatic external defibrillators may improve emergency heart care

Use of automatic external defibrillators (AEDs) by emergency medical personnel may facilitate treatment for more victims of heart attacks, according to a study reported in the *Journal of the American Medical Association*. The study found that automatic devices required less training for proper use and that they delivered shock to the heart one minute faster than standard defibrillators.

Richard O. Cummins, MD, MPH, MSc, of the University of Washington, Seattle, and colleagues compared the effectiveness of both kinds of defibrillators used by emergency medical technicians (EMTs) in treating 321 patients with cardiac arrest. In their study, 116 patients were treated with AEDs, and 158 were treated with standard defibrillators (an additional 47 were treated by EMTs using the standard defibrillator even though they were assigned to use the AED).

Hospital admission and discharge rates of patients were comparable regardless of the type of device used. "The only significant difference observed was in the time from power on to first shock: 1.1 minutes average auto group and 2.0 minutes average standard group," the researchers say.

"Automatic external defibrillators appear to have advantages over standard defibrillators in training, skill retention, and faster operation," the report adds, noting that AEDs can make early defibrillation available for a much larger portion of the population. The automatic devices take responsibility for rhythm identification away from prehospital rescuers, which can be beneficial especially in rural areas, where volunteers are trained to respond to emergencies.


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# Haines defends stand on pharmaceutical representatives

*To the Editor:* The finest compliment that a writer can hope for is to have his work read and to have it arouse strong reaction. I certainly seem to have succeeded in my December article on pharmaceutical representatives [JOSMA, Dec 86]. Unfortunately, all the reaction received by the JOURNAL was negative. I did receive several letters and personal communications strongly in agreement with my essay on drug reps. One colleague's letter read simply, "Regarding your article, 'The Ubiquitous Pharmaceutical Representative' . . . HOORAY!"

I did respond to each letter that I received and appreciate the opportunity to respond to my three critics in the March JOURNAL.

Dr. Tisdal accuses me of promoting unemployment when no reference to this topic was made. I did suggest that the drug reps move on to more satisfying occupations where society could make better use of their skills. Perhaps we could send some of our drug reps in the eastern part of the state out west to allow Dr. Tisdal a greater opportunity to trade hunting and fishing stories. I must admit that I was somewhat disappointed to see that he wanted my article "trashed." For an individual so tolerant of drug reps he doesn't seem tolerant of opinions that differ with his own.

Dr. Smithson claims that, "I wish to do nothing but read medical journals," an observation that I found both amusing and puzzling since no reference to this idea was made in my essay. What I do wish is to see my patients without being interrupted by salesmen. And no, I do not consider pharmaceutical booths at conventions the work of the devil; they are there for one purpose, namely to promote products that will hopefully make millons for the big drug companies.

Dr. Smithson seems to feel that medical meetings are dependent on the drug company support and this is exactly what they want him to believe. Our scientific meetings have been converted into circuses with innumerable sideshows where some salesman is hawking his wares. I see no need for physicians to feel that they must provide a forum for the drug companies every time they convene.

Dr. Confer's comments were also appreciated. He was upset because my article was one-sided. This comment was somewhat confusing since I wrote an opinion piece with a point of view and had no intention of attempting to present a balanced view of the topic. As far as the old argument that drug reps donate medications for our indigent patients, I have a ready response. Perhaps our poor and even our middle class patients could better afford their prescriptions if the drug companies did not pass along to the consumer their expenses which include the millions upon millions of dollars of advertising, drug rep salaries, "free lunches," cruises in the Caribbean, etc. I'm sure the patient's cost for medications would be a fraction of the current amount once all this waste was eliminated.

I think that the drug reps take advantage of our human nature by making us think that we are getting something for nothing. But what we are surrendering to them is our time, our most valuable (albeit fleeting) possession. And ultimately, time is all we really have anyway.

*Joe D. Haines, Jr., MD*
*Skiatook*

**June 1**
**is the closing date**
**for the**
**July 1987 JOURNAL**

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## Deaths

### Donald J. Blair
### 1929 - 1987

Donald J. Blair, OSMA executive director from 1962 to 1976, died March 16, 1987. A 1953 graduate of the University of Oklahoma, he became associate director at OSMA in 1955. He left OSMA in 1976 to become a vice president at C. L. Frates & Company, where he helped organize and manage three professional liability insurance companies, including PLICO. At the time of his death, he was executive director of Oklahomans Against Lawsuit Abuse. The JOURNAL will publish a tribute to Mr Blair in a forthcoming issue.

### Eldon Clyde Mohler, MD
### 1907 - 1987

Eldon C. Mohler, MD, 1958-59 OSMA president, died March 21, 1987. A Life Member and former member of the OSMA Board of Trustees, Dr Mohler retired in 1986 after 46 years of practice in Ponca City. He was born in Schell, Mo, and graduated from Northwestern University Medical School, Chicago, in 1939. Dr Mohler specialized in internal medicine and cardiovascular disease.

## In Memorium

### 1986

| | |
|---|---|
| *Marianne Elsbeth Kosbab, MD* | *June 13* |
| *William W. Rucks, Jr., MD* | *June 27* |
| *Ralph A. Smith, MD* | *July 27* |
| *Howard D. Tuttle, MD* | *August 3* |
| *Welborn W. Sanger, MD* | *September 19* |
| *William Carl Ewell, MD* | *September 20* |
| *Marcella Steel, MD* | *October 1* |
| *Terry Dwight Leming, MD* | *October 13* |
| *William Pat Fite, Jr., MD* | *October 30* |
| *Samuel Jackson McDaniel, MD* | *November 2* |
| *Iron Hawthorne Nelson, MD* | *November 12* |
| *John Robert Walter Spencer, MD* | *December 4* |

### 1987

| | |
|---|---|
| *Edward Leon Moore, MD* | *February 14* |
| *Ralph Cameron Emmott, MD* | *February 16* |
| *James Laurel Haddock, Jr., MD* | *February 19* |
| *Donald J. Blair* | *March 16* |
| *Eldon Clyde Mohler, MD* | *March 21* |

## Miscellaneous Advertisements

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In writing your ad, remember that it will be read statewide; include complete address and/or telephone information. If discussing employment, be sure to specify whether you are seeking a position or trying to fill one.

Enclose payment with your ad and mail to: OSMA JOURNAL, 601 Northwest Expressway, Oklahoma City, OK 73118. OSMA members and state agencies will be invoiced upon request.

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FOR SALE — 4,000-SQUARE-FOOT PHYSICIAN'S OFfice building, 15 years old, one story, built by Marshall Erdman, and located across the street from the community's hospital, in N.E. Oklahoma town of 17,000 serving a population of more than 45,000. The area is in need of OB-GYN (1 in town), GP-FP (3 in town), INT. MED. (3 going on 2 in town), and GEN. SURG. (2 in town). We have full time, board certified ORTHO., UROL., RAD. (CAT scan, ultrasound, nuc. med.) PED., PATH., and 24-hr. E.R. coverage. Available also, are one day a week consultation opportunities with Tulsa-based PUL. MED., CARD., ENT, ALLER., and G.I. specialists. The hospital has 4 O.R.s and a cysto room, 2 delivery rooms, newborn nursery, and a 9-bed med-surg I.C.U. Lots of good opportunities here. Contact Robert L. Alexander, M.D., 1212 S. Belmont, Okmulgee, OK 74447. Phone (918) 756-5471 or (918) 756-8313 after hours.

*(continued on p 327)*

# Motrin® 800 mg TABLETS

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J-6138 January 1986

## News from DISTA about a new dosage form of cephalexin

## ANNOUNCING NEW

# Keflet™ TABLETS

# cephalexin

## All the advantages of cephalexin in a convenient tablet form

- Backed by over 15 years of clinical experience
- Smaller tablet is specially shaped and coated for easier swallowing
- May enhance patient compliance, particularly among the elderly
- Tablet dosage form may be appreciated by patients of all ages

## NEW Keflet Tablets are available as:

250-mg Tablets

500-mg Tablets

Keflet is contraindicated in patients with known allergy to the cephalosporins and should be given cautiously to penicillin-sensitive patients.

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**Brief Summary. Consult the package literature for prescribing information.**
**Indications and Usage:** Keflet™ Tablets (cephalexin, Dista) are indicated for the treatment of the following infections when caused by susceptible strains of the designated microorganisms.

Respiratory tract infections caused by *Streptococcus pneumoniae* and group A β-hemolytic streptococci (Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. Keflet is generally effective in the eradication of streptococci from the nasopharynx; however, substantial data establishing the efficacy of Keflet in the subsequent prevention of rheumatic fever are not available at present.)

Otitis media due to *S pneumoniae, Haemophilus influenzae,* staphylococci, streptococci, and *Neisseria catarrhalis*

Skin and skin-structure infections caused by staphylococci and/or streptococci

Bone infections caused by staphylococci and/or *Proteus mirabilis*

Genitourinary tract infections, including acute prostatitis, caused by *Escherichia coli, P mirabilis,* and *Klebsiella* sp

*Note*—Culture and susceptibility tests should be initiated prior to and during therapy. Renal function studies should be performed when indicated.

**Contraindication:** Keflet is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

**Warnings:** BEFORE CEPHALEXIN THERAPY IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS AND PENICILLIN. CEPHALOSPORIN C DERIVATIVES SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN SENSITIVE PATIENTS.

SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES.

There is some clinical and laboratory evidence of partial cross-allergenicity of the penicillins and the cephalosporins. Patients have been reported to have had severe reactions (including anaphylaxis) to both drugs.

Any patient who has demonstrated some form of allergy, particularly to drugs, should receive antibiotics cautiously. No exception should be made with regard to Keflet.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics (including macrolides, semisynthetic penicillins, and cephalosporins), therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with the use of antibiotics. Such colitis may range in severity from mild to life-threatening.

Treatment with broad-spectrum antibiotics alters the normal flora of the colon and may permit overgrowth of clostridia. Studies indicate that a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis.

Mild cases of pseudomembranous colitis usually respond to drug discontinuance alone. In moderate to severe cases, management should include sigmoidoscopy, appropriate bacteriologic studies, and fluid, electrolyte, and protein supplementation. When the colitis does not improve after the drug has been discontinued, or when it is severe, oral vancomycin is the drug of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should be ruled out.

*Usage in Pregnancy*—Safety of this product for use during pregnancy has not been established.

**Precautions:** *General*—Patients should be followed carefully so that any side effects or unusual manifestations of drug idiosyncrasy may be detected. If an allergic reaction to Keflet occurs, the drug should be discontinued and the patient treated with the usual agents (eg, epinephrine or other pressor amines, antihistamines, or corticosteroids).

Prolonged use of Keflet may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs' tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs' testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs' test may be due to the drug.

Keflet should be administered with caution in the presence of markedly impaired renal function. Under such conditions, careful clinical observation and laboratory studies should be made because safe dosage may be lower than that usually recommended.

Indicated surgical procedures should be performed in conjunction with antibiotic therapy.

As a result of administration of Keflet, a false-positive reaction for glucose in the urine may occur. This has been observed with Benedict's and Fehling's solutions and also with Clinitest® tablets but not with Tes-Tape™ (Glucose Enzymatic Test Strip, USP, Lilly).

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

*Usage in Pregnancy—Pregnancy Category B*—The daily oral administration of cephalexin to rats in doses of 250 or 500 mg/kg prior to and during pregnancy, or to rats and mice during the period of organogenesis only, had no adverse effect on fertility, fetal viability, fetal weight, or litter size. Note that the safety of cephalexin during pregnancy in humans has not been established.

Cephalexin showed no enhanced toxicity in weanling and newborn rats as compared with adult animals. Nevertheless, because the studies in humans cannot rule out the possibility of harm, Keflet should be used during pregnancy only if clearly needed.

*Nursing Mothers*—The excretion of cephalexin in the milk increased up to 4 hours after a 500-mg dose, the drug reached a maximum level of 4 μg/mL, then decreased gradually, and had disappeared 8 hours after administration. Caution should be exercised when Keflet is administered to a nursing woman.

**Adverse Reactions:** *Gastrointestinal*—Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment. Nausea and vomiting have been reported rarely. The most frequent side effect has been diarrhea. It was very rarely severe enough to warrant cessation of therapy. Dyspepsia and abdominal pain have also occurred. As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.

*Hypersensitivity*—Allergic reactions in the form of rash, urticaria, angioedema, and, rarely, erythema multiforme, Stevens-Johnson Syndrome, or toxic epidermal necrolysis have been observed. These reactions usually subsided upon discontinuation of the drug. Anaphylaxis has also been reported.

Other reactions have included genital and anal pruritus, genital moniliasis, vaginitis and vaginal discharge, dizziness, fatigue, and headache. Eosinophilia, neutropenia, thrombocytopenia, and slight elevations in SGOT and SGPT have been reported.

Pv 2211 DPP

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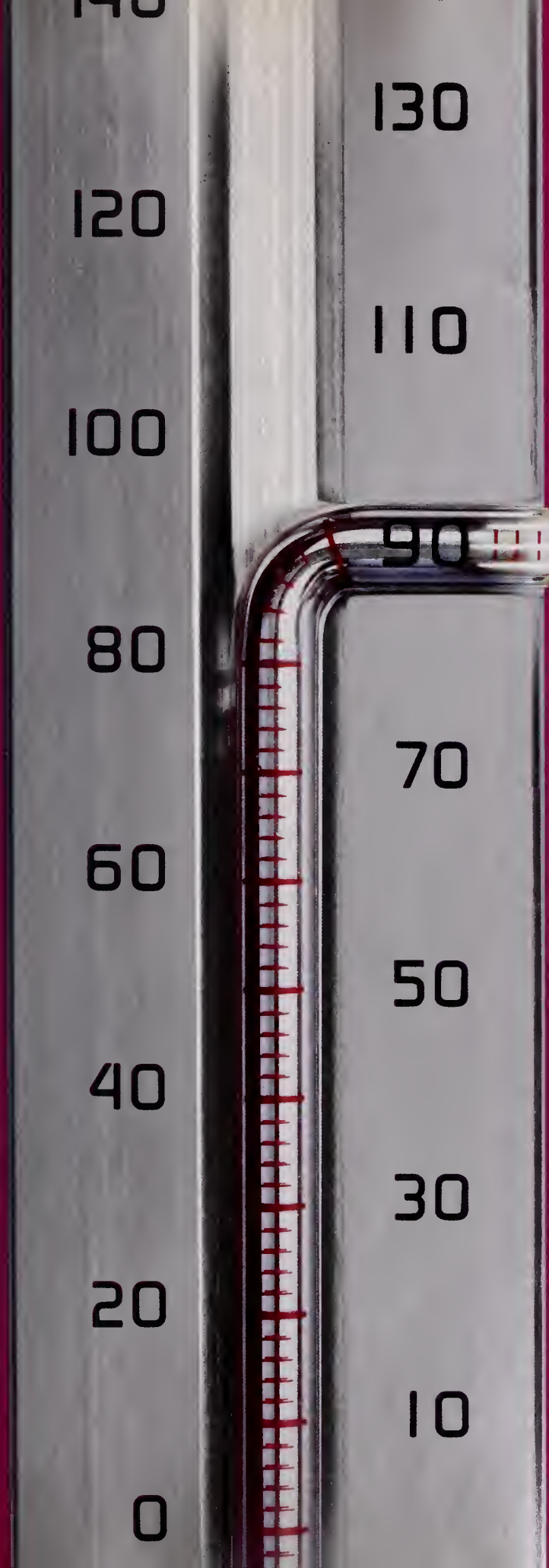


In mild to moderate hypertension

# THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER...

**NEW**
ONCE DAILY

# ISOPTIN SR*
## (verapamil HCl/Knoll)

***240 mg scored, sustained-release tablets***

**JAMES B.**
38, black male, heavy smoker. Prescribed a diuretic by another physician last year for hypertension.

**YOUR CONCERNS**
Presents with "smoker's cough." Workup reveals a BP of 150/107.

**A LOGICAL CHOICE FOR CONTROL OF HIS BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Black hypertensives often have low plasma renin activity and generally do not respond favorably to beta blockers.
— Beta blockers may increase the likelihood of bronchospasm.

**ALICE W.**
65, diabetic, overweight. Her BP has elevated to 190/98.

**YOUR CONCERNS**
She's on daily insulin.

**A LOGICAL CHOICE FOR CONTROL OF HER BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Unlike most beta blockers and diuretics, ISOPTIN has no adverse effects on serum glucose levels.
— Unlike most beta blockers, ISOPTIN does not mask the symptoms of hypoglycemia.

**THOMAS G.**
70, asthmatic. In the past, BP adequately controlled with 25 mg hydrochlorothiazide daily.

**YOUR CONCERNS**
Today patient presents with symptoms of gout. Workup reveals high uric acid level, low serum potassium, and BP elevated to 180/98.

**A LOGICAL CHOICE FOR CONTROL OF HIS BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Unlike diuretics, ISOPTIN will not decrease serum potassium levels or elevate uric acid levels.
— Unlike beta blockers, ISOPTIN can be used safely in asthma and COPD patients.

**JOHN K.**
42, Annual physical uncovered diastolic BP of 102... confirmed on three successive office visits. Unresponsive to nonpharmacologic intervention.

**YOUR CONCERNS**
Salesman, spends many hours of his working day in car... total cholesterol level 300, HDL 35.

**A LOGICAL CHOICE FOR CONTROL OF HIS BP**
ISOPTIN® (verapamil HCl/Knoll) because...
— Unlike diuretics, ISOPTIN does not cause urinary urgency.
— Unlike either beta blockers or diuretics, ISOPTIN will not adversely affect his already seriously compromised lipid profile.
— Unlike with propranolol, fatigue and impotence are rarely reported.

## *Antihypertensive therapy you and your patients can live with*

*A product of Knoll research.

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Please see adjacent page for brief summary.

*In mild to moderate hypertension* Brief Summary

***THE FIRST ONCE DAILY CALCIUM CHANNEL BLOCKER***

**ISOPTIN® SR (verapamil HCl/Knoll) 240 mg scored, sustained-release tablets**

**CONTRAINDICATIONS:** 1) Severe left ventricular dysfunction (see WARNINGS), 2) Hypotension (less than 90 mmHg systolic pressure) or cardiogenic shock, 3) Sick sinus syndrome or 2nd or 3rd degree AV block (except in patients with a functioning artificial ventricular pacemaker).

**WARNINGS: Heart Failure:** ISOPTIN should be avoided in patients with severe left ventricular dysfunction (see DRUG INTERACTIONS). Patients with milder ventricular dysfunction should, if possible, be controlled before verapamil treatment. Hypotension: ISOPTIN (verapamil HCl) may produce occasional symptomatic hypotension. Elevated Liver Enzymes: Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Periodic monitoring of liver function in patients receiving verapamil is therefore prudent. Accessory Bypass Tract (Wolff-Parkinson-White): Patients with paroxysmal and/or chronic atrial flutter or atrial fibrillation and a coexisting accessory AV pathway have developed increased antegrade conduction across the accessory pathway producing a very rapid ventricular response or ventricular fibrillation after receiving intravenous verapamil. While this has not been reported with oral verapamil, it should be considered a potential risk. Treatment is usually D.C.-cardioversion. Atrioventricular Block: The effect of verapamil on AV conduction and the SA node may cause asymptomatic 1st degree AV block and transient bradycardia. Higher degrees of AV block, while infrequent (0.8%), may require a reduction in dosage or, in rare instances, discontinuation of verapamil HCl. Patients with Hypertrophic Cardiomyopathy (IHSS): Although verapamil has been used in the therapy of patients with IHSS, severe cardiovascular decompensation and death have been noted in this patient population.

**PRECAUTIONS: Impaired Hepatic or Renal Function:** Verapamil is highly metabolized by the liver with about 70% of an administered dose excreted in the urine. In patients with impaired hepatic or renal function verapamil should be administered cautiously and the patients monitored for abnormal prolongation of the PR interval or other signs of excessive pharmacological effects (see OVERDOSAGE).

**Drug Interactions:** Beta Blockers: Concomitant use of ISOPTIN and oral beta-adrenergic blocking agents may be beneficial in certain patients with chronic stable angina or hypertension, but available information is not sufficient to predict with confidence the effects of concurrent treatment in patients with left ventricular dysfunction or cardiac conduction abnormalities. Digitalis: Clinical use of verapamil in digitalized patients has shown the combination to be well tolerated if digoxin doses are properly adjusted. However, chronic verapamil treatment increases serum digoxin levels by 50 to 75% during the first week of therapy and this can result in digitalis toxicity. Upon discontinuation of ISOPTIN (verapamil HCl), the patient should be reassessed to avoid underdigitalization. Antihypertensive Agents: Verapamil administered concomitantly with oral antihypertensive agents (e.g., vasodilators, angiotensin-converting enzyme inhibitors, diuretics, beta blockers, prazosin) will usually have an additive effect on lowering blood pressure. Patients receiving these combinations should be appropriately monitored. Disopyramide: Disopyramide should not be administered within 48 hours before or 24 hours after verapamil administration. Quinidine: In patients with hypertrophic cardiomyopathy (IHSS), concomitant use of verapamil and quinidine resulted in significant hypotension. There has been a report of increased quinidine levels during verapamil therapy. Nitrates: The pharmacologic profile of verapamil and nitrates as well as clinical experience suggest beneficial interactions. Cimetidine: Two clinical trials have shown a lack of significant verapamil interaction with cimetidine. A third study showed cimetidine reduced verapamil clearance and increased elimination to 1/2. Anesthetic Agents: Verapamil may potentiate the activity of neuromuscular blocking agents and inhalation anesthetics. Carbamazepine: Verapamil may increase carbamazepine concentrations during combined therapy. Rifampin: Therapy with rifampin may markedly reduce oral verapamil bioavailability. Lithium: Verapamil may lower lithium levels in patient on chronic oral lithium therapy. Carcinogenesis, Mutagenesis, Impairment of Fertility: There was no evidence of a carcinogenic potential of verapamil administered to rats for two years. Verapamil was not mutagenic in the Ames test. Studies in female rats did not show impaired fertility. Effects on male fertility have not been determined. Pregnancy (Category C): There are no adequate and well-controlled studies in pregnant women. ISOPTIN crosses the placental barrier and can be detected in umbilical vein blood at delivery. This drug should be used during pregnancy, labor, and delivery, only if clearly needed. Nursing Mothers: ISOPTIN is excreted in human milk, therefore, nursing should be discontinued while verapamil is administered. Pediatric Use: Safety and efficacy of ISOPTIN in children below the age of 18 years have not been established.

**ADVERSE REACTIONS:** Constipation 8.4%, dizziness 3.5%, nausea 2.7%, hypotension 2.5%, edema 2.1%, headache 1.9%, CHF/pulmonary edema 1.8%, fatigue 1.7%, bradycardia 1.4%, 3° AV block 0.8%, flushing 0.1%, elevated liver enzymes (see WARNINGS). The following reactions, reported in less than 1.0% of patients, occurred under conditions (open trials, marketing experience) where a causal relationship is uncertain; they are mentioned to alert the physician to a possible relationship: angina pectoris, arthralgia and rash, AV block, blurred vision, cerebrovascular accident, chest pain, claudication, confusion, diarrhea, dry mouth, dyspnea, ecchymosis or bruising, equilibrium disorders, exanthema, gastrointestinal distress, gingival hyperplasia, gynecomastia, hair loss, hyperkeratosis, impotence, increased urination, insomnia, macules, muscle cramps, myocardial infarction, palpitations, paresthesia, psychotic symptoms, purpura (vasculitis), shakiness, somnolence, spotty menstruation, sweating, syncope, urticaria. Treatment of Acute Cardiovascular Adverse Reactions: Whenever severe hypotension or complete AV block occur following oral administration of verapamil, the appropriate emergency measures should be applied immediately, e.g., intravenously administered isoproterenol HCl, levarterenol bitartrate, atropine (all in the usual doses), or calcium gluconate (10% solution). If further support is necessary, inotropic agents (dopamine or dobutamine) may be administered. Actual treatment and dosage should depend on the severity and the clinical situation and the judgment and experience of the treating physician.

**OVERDOSAGE:** Treatment of overdosage should be supportive. Beta-adrenergic stimulation or parenteral administration of calcium solutions may increase calcium ion flux across the slow channel, and have been used effectively in treatment of deliberate overdosage with verapamil. Clinically significant hypotensive reactions or fixed high degree AV block should be treated with vasopressor agents or cardiac pacing, respectively. Asystole should be handled by the usual measures including cardiopulmonary resuscitation.

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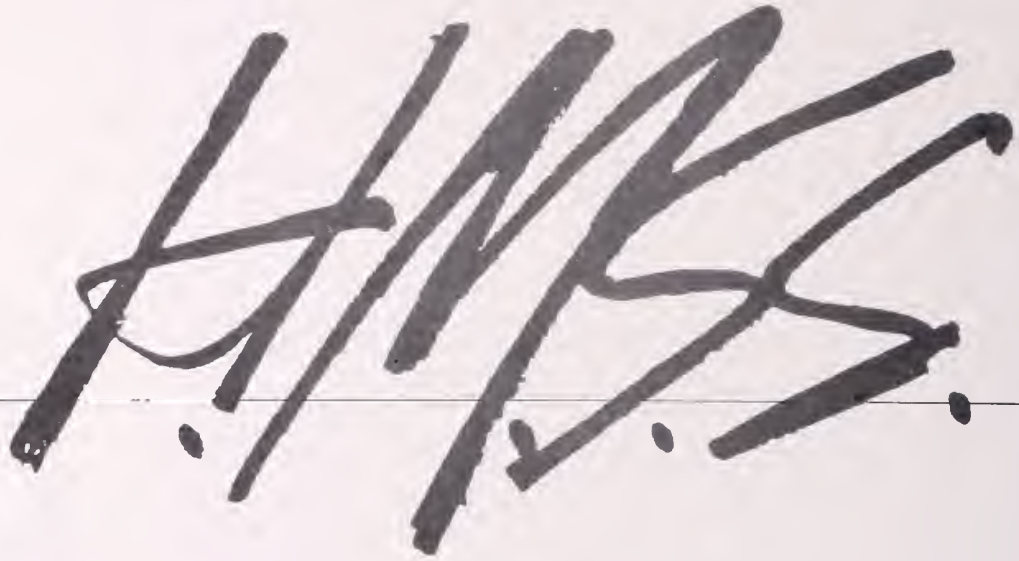

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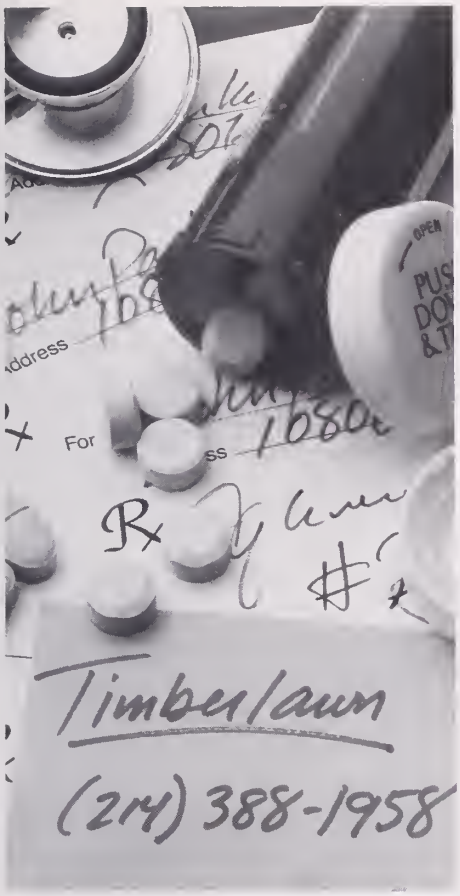


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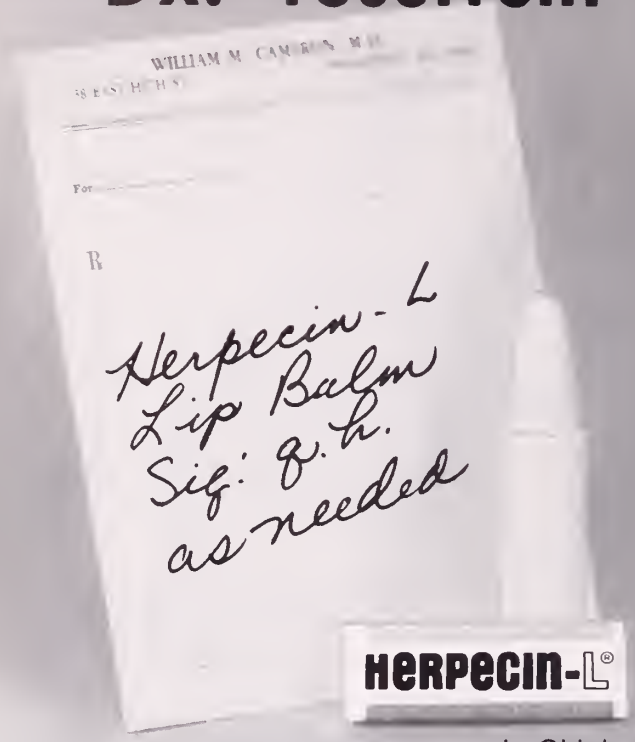


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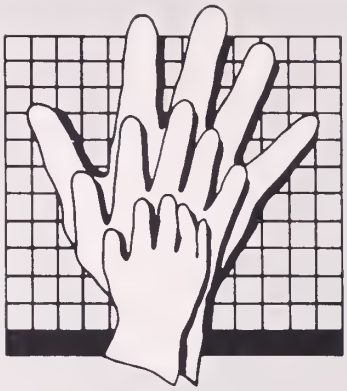

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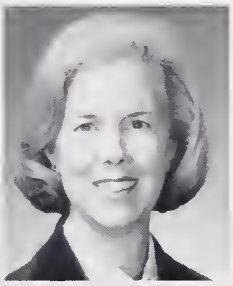

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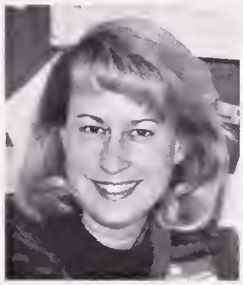

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■ **Charles W. Johnston, former vice president** for government relations for the Oklahoma Hospital Association, is the new director of corporate relations for Unisys Corporation in Oklahoma City. A former consultant for the Oklahoma State Medical Association, Johnston will be working with healthcare associations, providers, and the Oklahoma state legislature. Hospitals and physicians having problems with Medicaid claims processing should contact him at Unisys, Suite 66, 4545 North Lincoln Boulevard, Oklahoma City, OK 73105, (405) 528-7002.

■ **The American Medical Association's Con**ference on the Impaired Health Professional will be held Thursday through Saturday, October 8-11, 1987, at the Drake Hotel in Chicago. The major theme of this year's conference, "Impaired Health Professionals: Educating Ourselves . . . Educating Others," will stress the need for increased educational activities in the area of professional health impairment. A special feature of the conference will be the presentation of original research on topics related to impairment and well-being. Abstracts are now being sought; deadline for submission of research abstracts is August 1. For further information, contact Janice J. Robertson, AMA Department of Substance Abuse, 535 North Dearborn, Chicago, IL 60610, (312) 645-5083.

■ **The newly revised and expanded 7th edition** of *USP DI* (1987) is available from the United States Pharmacopeial Convention. *USP DI* is a comprehensive, continuously updated reference of drug use information for the health care professional and patient. It is used as the data base for the patient education leaflet programs of the American Medical Association, National Association of Retail Druggists, and American Academy of Family Physicians, among others. For information write to the USP Professional Affairs Department, 12601 Twinbrook Parkway, Rockville, MD 20852.

■ **Hospitals have a strong financial incen**tive to prevent nosocomial (hospital-acquired) infections under the prospective payment reimbursement system, says a study in the *Journal of the American Medical Association*. If this system, based on diagnosis related groups (DRGs), provided hospitals with substantial extra payments for each infectious complication, administrators might be tempted to reduce infection control efforts, Robert W. Haley, MD, of the University of Texas Health Science Center, Dallas, and colleagues say. However, they report, an analysis of some 9,500 hospital-acquired infections showed only a small percentage would have resulted in a higher DRG payment, which, when averaged out over all the infections studied, would cover only 5% of the hospitals' costs for treating the problems. "Thus," the study concludes, "at least 95% of the cost savings obtained from preventing nosocomial infections represents financial gains to the hospital."

■ **The CME program of the University of** Oklahoma College of Medicine will present the Department of Orthopaedic Surgery and Rehabilitation Annual Visiting Professorship Wednesday through Saturday, June 17-20, 1987. The objectives of the course are to review and discuss the contemporary experience of the department with a variety of orthopedic problems and to provide an opportunity for the exchange of ideas between the faculty and visitors. For information contact Magdalen De Bault, Associate Director, CME, OU College of Medicine, PO Box 26901, Room 164E, LB, Oklahoma City, OK 73190.

■ **It is not uncommon for critically ill patients** to require respirators for long periods of time, but little is known about the outcome of mechanical ventilation, in patients placed on such devices for more than a few days. A study in the March *Archives of Internal Medicine* finds prolonged mechanical ventilation is associated with limited survival and poor functional status in those who do survive. James E. Spicher, MD, and David P. White, MD, of the Pennsylvania State University's Milton S. Hershey Medical Center, Hershey, Pa, reviewed the medical records of 250 patients who spent at least 10 days on a respirator during a five-year period. Overall survival was 39% at discharge, 29% at one year, and 22.5% at two years. Age and functional status prior to respiratory failure were the best predictors of survival, the study says. Of patients who survived to discharge, 40% went to nursing homes and 33% were confined to their homes.

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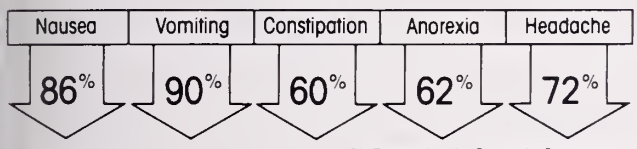


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Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage, withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline, symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
**Precautions:** Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady state concentrations of the tricyclic drugs. Concomitant use of Limbitrol with other psychotropic drugs has not been evaluated, sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration at ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
**Adverse Reactions:** Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
The following list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs.
*Cardiovascular:* Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
*Psychiatric:* Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
*Neurologic:* Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
*Anticholinergic:* Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
*Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
*Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
*Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
*Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion.
*Other:* Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
**Overdosage:** Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
**Dosage:** Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion at daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly. Limbitrol DS (double strength) Tablets, initial dosage of three or four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol Tablets, initial dosage of three or four tablets daily in divided doses, for patients who do not tolerate higher doses.
**How Supplied:** *Double strength (DS) Tablets*, white, film-coated, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt), and *Tablets*, blue, film-coated, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt). Available in bottles of 100 and 500, Tel-E-Dose® packages of 100, Prescription Paks of 50.

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## OKLAHOMA STATE MEDICAL ASSOCIATION

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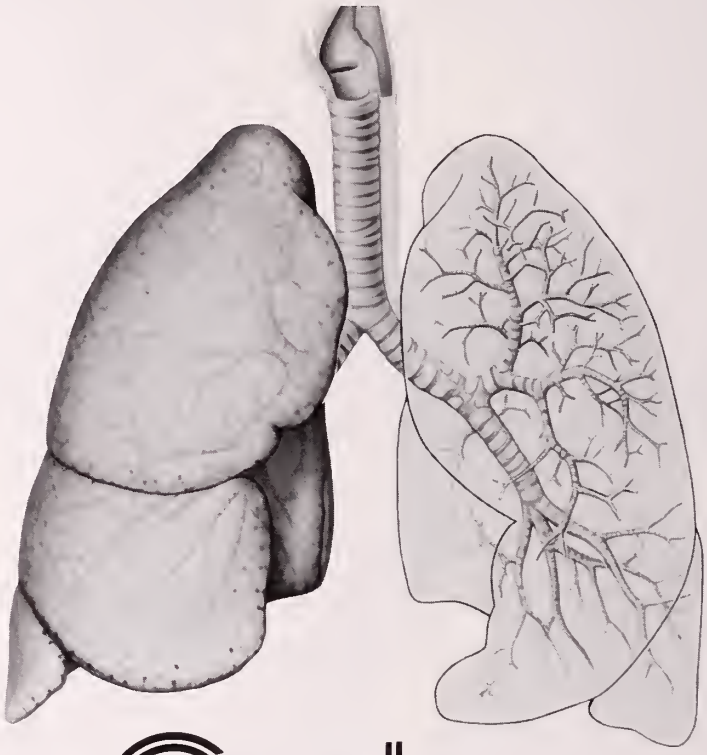

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Penicillin is the usual drug of choice in the treatment and prevention of streptococcal infections, including the prophylaxis of rheumatic fever. See prescribing information.

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**Summary. Consult the package literature for prescribing information.**

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**Contraindication:**
Known allergy to cephalosporins.

**Warnings:**
CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

**Precautions:**
- Discontinue Ceclor in the event of allergic reactions to it.
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- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

**Adverse Reactions:** (percentage of patients)
Therapy-related adverse reactions are uncommon. Those reported include:
- Gastrointestinal (mostly diarrhea): 2.5%.
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] or the above skin manifestations accompanied by arthritis/arthralgia and, frequently, fever): 1.5%; usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1%; and, rarely, thrombocytopenia.

**Abnormalities in laboratory results of uncertain etiology**
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

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# Freedom from pain

## Just one part of pain relief therapy.

### Vicodin® provides greater patient acceptance

**COMPARATIVE PHARMACOLOGY OF THREE ANALGESICS**

| | CONSTIPATION | RESPIRATORY DEPRESSION | SEDATION | EMESIS | PHYSICAL DEPENDENCE |
|---|---|---|---|---|---|
| HYDROCODONE | | X | | | X |
| CODEINE | X | X | X | X | X |
| OXYCODONE | XX | XX | XX | XX | XX |

**Blank space indicates that no such activity has been reported.**

Table adapted from Facts and Comparisons (Nov.) 1984 and Catalano RB. The medical approach to management of pain caused by cancer. "Semin Oncol" 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. "Ann Intern Med" 1980; 93; 588-96.

- Vicodin offers: less nausea, less sedation, less constipation.

***...and longer lasting pain relief– up to 6 hours.***

- Vicodin contains hydrocodone not codeine. In one study, 10 mg. of hydrocodone alone was shown to be as effective as 60 mg. of codeine.[1]
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- Dosage flexibility–1 tablet every 6 hours or 2 tablets every 6 hours (up to 8 tablets in 24 hours).

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hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

***The original hydrocodone analgesic.***

# Specify "Dispense as written" for the original hydrocodone analgesic.

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**CONTRAINDICATIONS:** Hypersensitivity to acetaminophen or hydrocodone.
**WARNINGS:**
**Drug Abuse and Dependence:** VICODIN® is subject to the Federal Controlled Substances Act (Schedule III). Psychic dependence, physical dependence and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN should be prescribed and administered with the same caution appropriate to the use of other oral-narcotic-containing medications.
**Respiratory Depression:** At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression by acting directly on brain stem respiratory centers. Hydrocodone also affects centers that control respiratory rhythm, and may produce irregular and periodic breathing.
**Head Injury and Increased Intracranial Pressure:** The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.
**Acute Abdominal Conditions:** The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.
**PRECAUTIONS:**
**Special Risk Patients:** VICODIN should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.
**Information For Patients:** VICODIN, like all narcotics, may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery; patients should be cautioned accordingly.
**Cough Reflex:** Hydrocodone suppresses the cough reflex; caution should be exercised when VICODIN is used postoperatively and in patients with pulmonary disease.
**Drug Interactions:** The CNS-depressant effects of VICODIN may be additive with that of other CNS depressants. When combined therapy is contemplated, the dose of one or both agents should be reduced. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.
**Usage in Pregnancy:** Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.
**Nonteratogenic Effects:** Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The intensity of the syndrome does not always correlate with the duration of maternal opioid use or dose.
**Labor and Delivery:** Administration of VICODIN to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.
**Nursing Mothers:** It is not known whether this drug is excreted in human milk; therefore, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.
**Pediatric Use:** Safety and effectiveness in children have not been established.
**ADVERSE REACTIONS:**
**Central Nervous System:** Sedation, drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, dizziness, psychic dependence, mood changes.
**Gastrointestinal System:** Nausea and vomiting may occur; they are more frequent in ambulatory than in recumbent patients. Prolonged administration of VICODIN may produce constipation.
**Genitourinary System:** Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.
**Respiratory Depression:** (See WARNINGS.)
**DOSAGE AND ADMINISTRATION:** Dosage should be adjusted according to the severity of the pain and the response of the patient. However, tolerance to hydrocodone can develop with continued use, and the incidence of untoward effects is dose related.
The usual dose is one tablet every six hours as needed for pain. (If necessary, this dose may be repeated at four-hour intervals.) In cases of more severe pain, two tablets every six hours (up to eight tablets in 24 hours) may be required.

Revised, April 1982. 5685


1. Hopkinson JH III: *Curr Ther Res* 24: 503-516, 1978
2. Beaver, WT *Arch Intern Med,* 141:293-300, 1981.


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# vicodin® III

hydrocodone bitartrate 5 mg. (Warning: May be habit forming) with acetaminophen 500 mg.

Before prescribing, see complete prescribing information in SK&F CO. literature or *PDR*. The following is a brief summary.

* **WARNING**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

**Contraindications:** Concomitant use with other potassium-sparing agents such as spironolactone or amiloride. Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

**Warnings:** **Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired.** If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum $K^+$ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict $K^+$ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available. Sensitivity reactions may occur in patients with or without a history of allergy or bronchial asthma. Possible exacerbation or activation of systemic lupus erythematosus has been reported with thiazide diuretics.

**Precautions:** The bioavailability of the hydrochlorothiazide component of 'Dyazide' is about 50% of the bioavailability of the single entity. Theoretically, a patient transferred from the single entities of triamterene and hydrochlorothiazide may show an increase in blood pressure or fluid retention. Similarly, it is also possible that the lesser hydrochlorothiazide bioavailability could lead to increased serum potassium levels. However, extensive clinical experience with 'Dyazide' suggests that these conditions have not been commonly observed in clinical practice. Angiotensin-converting enzyme (ACE) inhibitors can elevate serum potassium; use with caution with 'Dyazide'. Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids, and during concurrent use with amphotericin B or corticosteroids or corticotropin [ACTH]). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Cumulative effects of the drug may develop in patients with impaired renal function. Thiazides should be used with caution in patients with impaired hepatic function. They can precipitate coma in patients with severe liver disease. Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic and hemolytic anemia have been reported with thiazides. Thiazides may cause manifestation of latent diabetes mellitus. The effects of oral anticoagulants may be decreased when used concurrently with hydrochlorothiazide; dosage adjustments may be necessary. Clinically insignificant reductions in arterial responsiveness to norepinephrine have been reported. Thiazides have also been shown to increase the paralyzing effect of nondepolarizing muscle relaxants such as tubocurarine. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effects may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. Triamterene has been found in renal stones in association with the other usual calculus components. Therefore, 'Dyazide' should be used with caution in patients with histories of stone formation. A few occurrences of acute renal failure have been reported in patients on 'Dyazide' when treated with indomethacin. Therefore, caution is advised in administering nonsteroidal anti-inflammatory agents with 'Dyazide'. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine. Hypokalemia is uncommon with 'Dyazide', but should it develop, corrective measures should be taken such as potassium supplementation or increased dietary intake of potassium-rich foods. Corrective measures should be instituted cautiously and serum potassium levels determined. Discontinue corrective measures and 'Dyazide' should laboratory values reveal elevated serum potassium. Chloride deficit may occur as well as dilutional hyponatremia. Concurrent use with chlorpropamide may increase the risk of severe hyponatremia. Serum PBI levels may decrease without signs of thyroid disturbance. Calcium excretion is decreased by thiazides. 'Dyazide' should be withdrawn before conducting tests for parathyroid function. Thiazides may add to or potentiate the action of other antihypertensive drugs. Diuretics reduce renal clearance of lithium and increase the risk of lithium toxicity.

**Adverse Reactions:** Muscle cramps, weakness, dizziness, headache, dry mouth; anaphylaxis, rash, urticaria, photosensitivity, purpura, other dermatological conditions; nausea and vomiting, diarrhea, constipation, other gastrointestinal disturbances; postural hypotension (may be aggravated by alcohol, barbiturates, or narcotics). Necrotizing vasculitis, paresthesias, icterus, pancreatitis, xanthopsia and respiratory distress including pneumonitis and pulmonary edema, transient blurred vision, sialadenitis, and vertigo have occurred with thiazides alone. Triamterene has been found in renal stones in association with other usual calculus components. Rare incidents of acute interstitial nephritis have been reported. Impotence has been reported in a few patients on 'Dyazide', although a causal relationship has not been established.

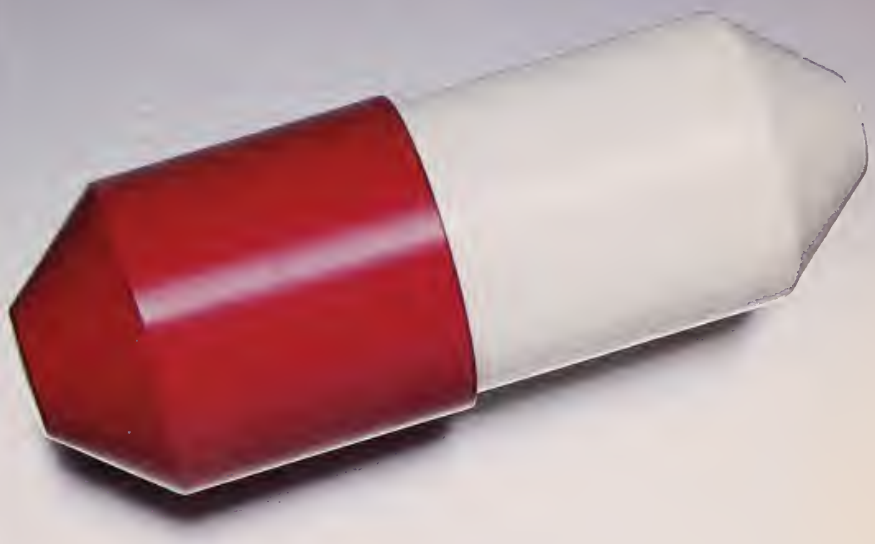
**Supplied:** **'Dyazide' is supplied as a red and white capsule, in bottles of 1000 capsules; Single Unit Packages (unit-dose) of 100 (intended for institutional use only); in Patient-Pak™ unit-of-use bottles of 100.**

BRS-DZ:L42

# In Hypertension*... When You Need to Conserve $K^+$

## Remember the Unique Red and White Capsule: Your Assurance of SK&F Quality

Serum $K^+$ and BUN should be checked periodically (see Warnings and Precautions).



Potassium-Sparing

**DYAZIDE®**

**25 mg** Hydrochlorothiazide/**50 mg** Triamterene/SKF

**Over 20 Years of Confidence**

The unique red and white Dyazide® capsule: Your assurance of SK&F quality.

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# Motrin® 800 TABLETS mg

ibuprofen

## Extra strength
## Convenience
## Economy

Upjohn

A Century of Caring
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J-6138 January 1986

# So my cousin lives next door to a lady who knows this man who overheard the teller at the bank say that he'd seen how much money Doctor...

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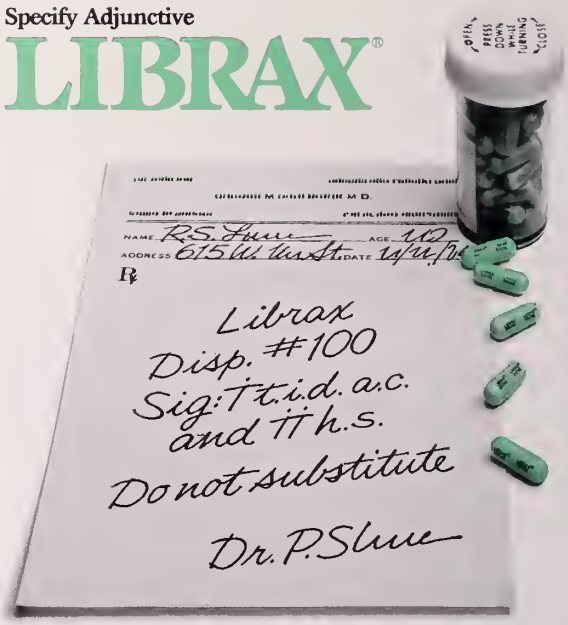
Specify Adjunctive

# LIBRAX®




**Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide**

**Please consult complete prescribing information, a summary of which follows:**

* **Indications:** Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: as adjunctive therapy in the treatment of peptic ulcer and in the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis. Final classification of the less-than-effective indications requires further investigation.

**Contraindications:** Glaucoma; prostatic hypertrophy, benign bladder neck obstruction; hypersensitivity to chlordiazepoxide HCl and/or clidinium Br.
**Warnings:** Caution patients about possible combined effects with alcohol and other CNS depressants, and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Physical and psychological dependence rarely reported on recommended doses, but use caution in administering Librium® (chlordiazepoxide HCl/Roche) to known addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions) reported following discontinuation of the drug.

*Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy. Advise patients to discuss therapy if they intend to or do become pregnant.

As with all anticholinergics, inhibition of lactation may occur.
**Precautions:** In elderly and debilitated, limit dosage to smallest effective amount to preclude ataxia, oversedation, confusion (no more than 2 capsules/day initially; increase gradually as needed and tolerated). Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider pharmacology of agents, particularly potentiating drugs such as MAO inhibitors, phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions reported in psychiatric patients. Employ usual precautions in treating anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship not established.
**Adverse Reactions:** No side effects or manifestations not seen with either compound alone reported with Librax. When chlordiazepoxide HCl is used alone, drowsiness, ataxia, confusion may occur, especially in elderly and debilitated; avoidable in most cases by proper dosage adjustment, but also occasionally observed at lower dosage ranges. Syncope reported in a few instances. Also encountered: isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent, generally controlled with dosage reduction; changes in EEG patterns may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice, hepatic dysfunction reported occasionally with chlordiazepoxide HCl, making periodic blood counts and liver function tests advisable during protracted therapy. Adverse effects reported with Librax typical of anticholinergic agents, *i.e.*, dryness of mouth, blurring of vision, urinary hesitancy, constipation. Constipation has occurred most often when Librax therapy is combined with other spasmolytics and/or low residue diets.

Roche Products Inc.
Manati, Puerto Rico 00701

P.I. 0186

To insist on the brand, be sure to write "Do Not Substitute" on your prescription.

When brain and bowel conflict...

# It's time for the Peacemaker.

In irritable bowel syndrome* anxiety can aggravate intestinal symptoms, which may further intensify anxiety — a distressing cycle of brain/bowel conflict. Librax intervenes with two well-known compounds. The Librium® (chlordiazepoxide HCl/Roche) component safely relieves anxiety. And Quarzan® (clidinium bromide/Roche) provides antisecretory and antispasmodic action to relieve discomfort associated with intestinal hypermotility.

Dual action — for peace between brain and bowel. Because of possible CNS effects, caution patients about engaging in activities requiring complete mental alertness. Specify Adjunctive

# LIBRAX®

Each capsule contains 5 mg chlordiazepoxide HCl and 2.5 mg clidinium bromide

*Librax has been evaluated as possibly effective as adjunctive therapy in the treatment of peptic ulcer and the irritable bowel syndrome.

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# LOSS PREVENTION SEMINAR ATTENDANCE MANDATORY

Attendance at an OSMA-PLICO sponsored Loss Prevention Seminar is now mandatory for all PLICO-insured physicians at least once every three years. **If a physician has never attended a seminar, he or she must attend one during 1987.** If a physician has not attended a program since 1984, he or she must attend this year, also.

The change making seminar attendance mandatory was implemented by the PLICO Board of Directors in late 1985, and a special endorsement outlining the requirement was included in all 1986 PLICO professional liability insurance policies. Any physician needing to attend in 1987, and failing to do so, will not be eligible for renewal of his or her PLICO professional liability insurance for calendar year 1988.

A Seminar Registration Form is located at the bottom of this page and a schedule of upcoming seminars is included. The registration form may be used for any seminar, but please **specify the date** you would like to attend. It is advisable to pre-register for the seminar you would like to attend.

## OSMA-PLICO
## 1987 LOSS PREVENTION SEMINARS

| | | |
|---|---|---|
| July 18 | Sat. 2-5 p.m. | LAWTON<br>Holiday Inn |
| August 8 | Sat. 2-5 p.m. | WOODWARD<br>Park Inn |
| September 19 | Sat. 8:30 a.m.-11:30 a.m. | McALESTER<br>Holiday Inn |
| September 26 | Sat. 8:30 a.m.-11:30 a.m. | OKLAHOMA CITY<br>Lincoln Plaza |

## LOSS PREVENTION SEMINAR REGISTRATION

Please register the following physician to attend the 1987 Loss Prevention Seminar indicated below (PLEASE TYPE OR PRINT):

Physician's Name: _____

Address: _____ City/State _____ Zip _____

Seminar Date and City: _____

Mail form to Oklahoma State Medical Association, 601 NW Expressway, Oklahoma City, OK 73118.

"Living in the city is lonely enough... with herpes it's like solitary confinement."
ZOVIRAX®
(acyclovir)
CAPSULES
Prevent genital herpes recurrences month after month with daily therapy.
(In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.)
Please see last page of this advertisement for brief summary of prescribing information.

# ZOVIRAX®
# (acyclovir)
# CAPSULES

## Help free your patients from recurrences.

## Daily therapy

Coping with genital herpes is rarely easy. For some, the worst part is the pain and discomfort of frequent attacks —month after month, year after year. For others, the emotional burden presents a more difficult problem, leading to social isolation, anxiety, and diminished self-esteem.

## Prevent or reduce recurrences

Although your patients have to live with herpes, they shouldn't have to suffer. Daily therapy with ZOVIRAX CAPSULES can help free them from the cycle of recurrent genital herpes. For many, one capsule three times a day can suppress recurrences completely while on therapy.

## Generally well tolerated

Daily therapy with ZOVIRAX CAPSULES is generally well tolerated. The most frequent adverse reactions reported during clinical trials were headache, diarrhea, nausea/vomiting, vertigo, and arthralgia.

The physical and emotional difficulties posed by genital herpes are unique for each patient. The frequency and severity of recurrent episodes, as well as the emotional impact of the disease, should be considered when selecting daily therapy with ZOVIRAX CAPSULES.

*Please see brief summary of prescribing information on next page.*

86-ZO

# Prevent recurrences month after month*

# ZOVIRAX® (acyclovir) CAPSULES

Brief Summary

**INDICATIONS AND USAGE:** Zovirax Capsules are indicated for the treatment of initial episodes and the management of recurrent episodes of genital herpes in certain patients.

The severity of disease is variable depending upon the immune status of the patient, the frequency and duration of episodes, and the degree of cutaneous or systemic involvement. These factors should determine patient management, which may include symptomatic support and counseling only, or the institution of specific therapy. The physical, emotional and psychosocial difficulties posed by herpes infections as well as the degree of debilitation, particularly in immunocompromised patients, are unique for each patient, and the physician should determine therapeutic alternatives based on his or her understanding of the individual patient's needs. Thus Zovirax Capsules are not appropriate in treating all genital herpes infections. The following guidelines may be useful in weighing the benefit/risk considerations in specific disease categories:

**First Episodes** (primary and nonprimary infections — commonly known as initial genital herpes):

Double-blind, placebo-controlled studies have demonstrated that orally administered Zovirax significantly reduced the duration of acute infection (detection of virus in lesions by tissue culture) and lesion healing. The duration of pain and new lesion formation was decreased in some patient groups. The promptness of initiation of therapy and/or the patient's prior exposure to Herpes simplex virus may influence the degree of benefit from therapy. Patients with mild disease may derive less benefit than those with more severe episodes. In patients with extremely severe episodes, in which prostration, central nervous system involvement, urinary retention or inability to take oral medication require hospitalization and more aggressive management, therapy may be best initiated with intravenous Zovirax.

**Recurrent Episodes:**

Double-blind, placebo-controlled studies in patients with frequent recurrences (6 or more episodes per year) have shown that Zovirax Capsules given for 4 to 6 months prevented or reduced the frequency and/or severity of recurrences in greater than 95% of patients. Clinical recurrences were prevented in 40 to 75% of patients. Some patients experienced increased severity of the first episode following cessation of therapy; the severity of subsequent episodes and the effect on the natural history of the disease are still under study.

The safety and efficacy of orally administered acyclovir in the suppression of frequent episodes of genital herpes have been established only for up to 6 months. Chronic suppressive therapy is most appropriate when, in the judgement of the physician, the benefits of such a regimen outweigh known or potential adverse effects. In general, Zovirax Capsules should not be used for the suppression of recurrent disease in mildly affected patients. Unanswered questions concerning the human relevance of *in vitro* mutagenicity studies and reproductive toxicity studies in animals given very high doses of acyclovir for short periods (see Carcinogenesis, Mutagenesis, Impairment of Fertility) should be borne in mind when designing long-term management for individual patients. Discussion of these issues with patients will provide them the opportunity to weigh the potential for toxicity against the severity of their disease. Thus, this regimen should be considered only for appropriate patients and only for six months until the results of ongoing studies allow a more precise evaluation of the benefit/risk assessment of prolonged therapy.

Limited studies have shown that there are certain patients for whom intermittent short-term treatment of recurrent episodes is effective. This approach may be more appropriate than a suppressive regimen in patients with infrequent recurrences.

Immunocompromised patients with recurrent herpes infections can be treated with either intermittent or chronic suppressive therapy. Clinically significant resistance, although rare, is more likely to be seen with prolonged or repeated therapy in severely immunocompromised patients with active lesions.

**CONTRAINDICATIONS:** Zovirax Capsules are contraindicated for patients who develop hypersensitivity or intolerance to the components of the formulation.

**WARNINGS:** Zovirax Capsules are intended for oral ingestion only.

**PRECAUTIONS: General:** Zovirax has caused decreased spermatogenesis at high doses in some animals and mutagenesis in some acute studies at high concentrations of drug (see PRECAUTIONS — Carcinogenesis, Mutagenesis, Impairment of Fertility). The recommended dosage and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION).

Exposure of Herpes simplex isolates to acyclovir *in vitro* can lead to the emergence of less sensitive viruses. The possibility of the appearance of less sensitive viruses in man must be borne in mind when treating patients. The relationship between the *in vitro* sensitivity of Herpes simplex virus to acyclovir and clinical response to therapy has yet to be established.

Because of the possibility that less sensitive virus may be selected in patients who are receiving acyclovir, all patients should be advised to take particular care to avoid potential transmission of virus if active lesions are present while they are on therapy. In severely immunocompromised patients, the physician should be aware that prolonged or repeated courses of acyclovir may result in selection of resistant viruses which may not fully respond to continued acyclovir therapy.

**Drug Interactions:** Co-administration of probenecid with intravenous acyclovir has been shown to increase the mean half-life and the area under the concentration-time curve. Urinary excretion and renal clearance were correspondingly reduced.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg given by gavage. There was no statistically significant difference in the incidence of tumors between treated and control animals, nor did acyclovir shorten the latency of tumors. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive life-time bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system considered less sensitive.

In acute studies, there was an increase, not statistically significant, in the incidence of chromosomal damage at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats but not Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found after 5 days dosing in a dominant lethal study in mice. In 6 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. At 3 loci in a Chinese hamster ovary cell line, the results were inconclusive. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive responses for mutagenicity and chromosomal damage occurred, but only at concentrations at least 400 times the acyclovir plasma levels achieved in man.

Acyclovir has not been shown to impair fertility or reproduction in mice (450 mg/kg/day, p.o.) or in rats (25 mg/kg/day, s.c.). At 50 mg/kg/day s.c. in the rat, there was a statistically significant increase in post-implantation loss, but no concomitant decrease in litter size. In female rabbits treated subcutaneously with acyclovir subsequent to mating, there was a statistically significant decrease in implantation efficiency but no concomitant decrease in litter size at a dose of 50 mg/kg/day. No effect upon implantation efficiency was observed when the same dose was administered intravenously. In a rat peri- and postnatal study at 50 mg/kg/day s.c., there was a statistically significant decrease in the group mean numbers of corpora lutea, total implantation sites and live fetuses in the $F_1$ generation. Although not statistically significant, there was also a dose related decrease in group mean numbers of live fetuses and implantation sites at 12.5 mg/kg/day and 25 mg/kg/day, s.c. The intravenous administration of 100 mg/kg/day, a dose known to cause obstructive nephropathy in rabbits, caused a significant increase in fetal resorptions and a corresponding decrease in litter size. However, at a maximum tolerated intravenous dose of 50 mg/kg/day in rabbits, there were no drug-related reproductive effects.

Intraperitoneal doses of 320 or 80 mg/kg/day acyclovir given to rats for 1 and 6 months, respectively, caused testicular atrophy. Testicular atrophy was persistent through the 4-week postdose recovery phase after 320 mg/kg/day; some evidence of recovery of sperm production was evident 30 days postdose. Intravenous doses of 100 and 200 mg/kg/day acyclovir given to dogs for 31 days caused aspermatogenesis. Testicles were normal in dogs given 50 mg/kg/day, i.v. for one month.

**Pregnancy:** ***Teratogenic Effects:*** Pregnancy Category C. Acyclovir was not teratogenic in the mouse (450 mg/kg/day, p.o.), rat (50 mg/kg/day, s.c.) or rabbit (50 mg/kg/day, s.c. and i.v.). There are no adequate and well-controlled studies in pregnant women. Acyclovir should not be used during pregnancy unless the potential benefit justifies the potential risk to the fetus. Although acyclovir was not teratogenic in animal studies, the drug's potential for causing chromosome breaks at high concentration should be taken into consideration in making this determination.

**Nursing Mothers:** It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zovirax is administered to a nursing woman. In nursing mothers, consideration should be given to not using acyclovir treatment or discontinuing breastfeeding.

**Pediatric Use:** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS — Short-Term Administration:** The most frequent adverse reactions reported during clinical trials were nausea and/or vomiting in 8 of 298 patient treatments (2.7%) and headache in 2 of 298 (0.6%). Less frequent adverse reactions, each of which occurred in 1 of 298 patient treatments (0.3%), included diarrhea, dizziness, anorexia, fatigue, edema, skin rash, leg pain, inguinal adenopathy, medication taste and sore throat.

**Long-Term Administration:** The most frequent adverse reactions reported in studies of daily therapy for 3 to 6 months were headache in 33 of 251 patients (13.1%), diarrhea in 22 of 251 (8.8%), nausea and/or vomiting in 20 of 251 (8.0%), vertigo in 9 of 251 (3.6%), and arthralgia in 9 of 251 (3.6%). Less frequent adverse reactions, each of which occurred in less than 3% of the 251 patients (see number of patients in parentheses), included skin rash (7), insomnia (4), fatigue (7), fever (4), palpitations (1), sore throat (2), superficial thrombophlebitis (1), muscle cramps (2), pars planitis (1), menstrual abnormality (4), acne (3), lymphadenopathy (2), irritability (1), accelerated hair loss (1), and depression (1).

**DOSAGE AND ADMINISTRATION: Treatment of initial genital herpes:** One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 10 days (total 50 capsules).

**Chronic suppressive therapy for recurrent disease:** One 200 mg capsule 3 times daily for up to 6 months. Some patients may require more drug, up to one 200 mg capsule 5 times daily for up to 6 months.

**Intermittent Therapy:** One 200 mg capsule every 4 hours, while awake, for a total of 5 capsules daily for 5 days (total 25 capsules). Therapy should be initiated at the earliest sign or symptom (prodrome) of recurrence.

**Patients With Acute or Chronic Renal Impairment:** One 200 mg capsule every 12 hours is recommended for patients with creatinine clearance $\leq$10 ml/min/1.73/m$^2$.

**HOW SUPPLIED:** Zovirax Capsules (blue, opaque) containing 200 mg acyclovir and printed with "Wellcome ZOVIRAX 200"- Bottles of 100 (NDC-0081-0991-55) and unit dose pack of 100 (NDC-0081-0991-56).

Store at 15°-30°C (59°-86°F) and protect from light.

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*In controlled studies, recurrences were totally prevented for 4 to 6 months in up to 75% of patients.

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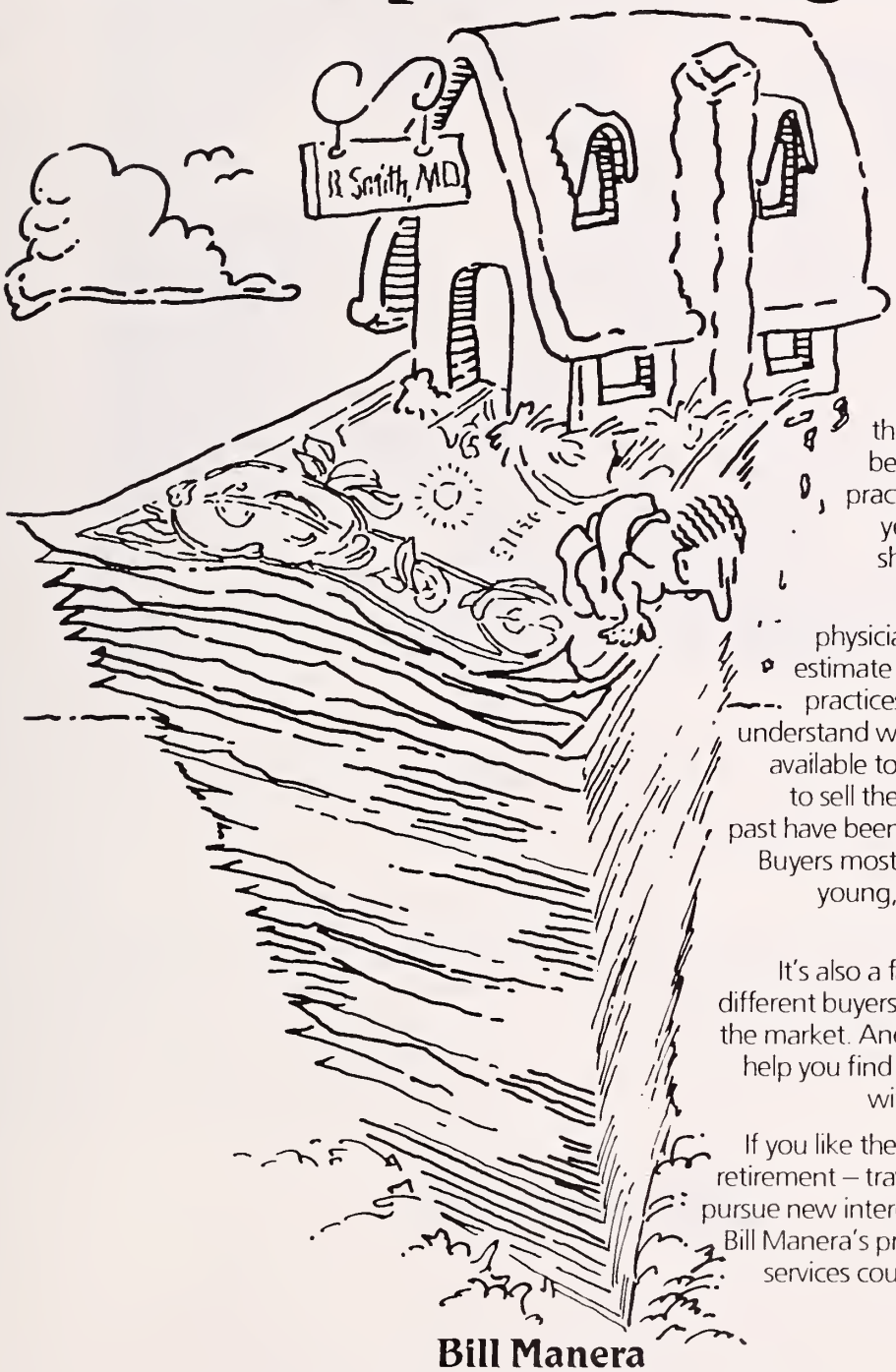
# Your practice may be worth much more than you thought.




If you have *ever considered* retirement, but thought you would be financially ahead practicing a few more years, perhaps you should *reconsider.*

The fact is, most physicians greatly underestimate the value of their practices. And it's easy to understand why. The networks available to physicians trying to sell their practices in the past have been mostly informal. Buyers most often have been young, with little capital to invest.

It's also a fact that new and different buyers are appearing in the market. And Bill Manera can help you find the right buyer – with the right price.

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## And she can with Allied Nursing Care.

You demand quality service for your patients and Allied Nursing Care provides the best care, anywhere in Oklahoma.

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**Allied Nursing Care is ready to serve . . . when she wants to go home.**

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(405) 848-1234

# New Ideas and New Ethics

I got a piece in the mail yesterday urging me to employ this outfit to show me how to build my practice. They promised to make me an expert in "attracting" patients to come see me and then use them to get more patients to come to me. They promised to let me in on the secrets of "effective marketing techniques, including advertising my practice in the graphic and electronic media." They assured me it would all be done in an "inoffensive, professional manner." Testimonials from many of my "own colleagues from coast to coast" were included. Seems these colleagues swore by this outfit. They had watched their practices grow way beyond their expectations in "an incredibly short time, in spite of the highly competitive atmosphere in today's marketplace!"

I got to thinking about their proposition. I didn't use their toll-free number to get more information or to invite one of their agents to visit me or find out how much their services would cost me, 'cause it just didn't make much sense to me, old timey as I am.

There I'd be, spending thousands of dollars to get more folks to leave their doctors and start coming to me. Or, if they didn't already have a doctor, or maybe even need one, to come see me.

"No, Doctor," I could imagine them saying, "I'm really in very good health, thanks to my regular doctor, but your ads were so convincing and your marketing techniques so effective I just couldn't resist making an appointment to see you."

Or maybe something like this: "Naw, I ain't got a regular doctor. I'm healthy as a horse 'cause I don't never go to doctors or take any medicine. I don't believe in doctorin'. Never been sick a day in my life 'cause I don't take no damn pills. I just come in to see ya 'cause your commercials said I ought to — and 'cause two of my best buddies was in last week."

I figured I didn't want to pay for any marketing techniques or advertising that was quite so successful. I figured there must be other ways to "build up" my "lagging practice and diminishing cash flow."

It wouldn't cost me anything to tell the few patients I have to quit exercising and start eating more cream gravy and pork chops. I could tell them that good food and pure salt never hurt anybody. I could put signs in my office which said "Thank you for smoking. You will need to see me more frequently. You will need to buy more medicine. You will be raising tax revenues, and you will be increasing my cash flow."

Talk about getting patients to refer other patients to me — a doctor who says it's OK to eat what you like, sit on your butt, and smoke! My God; I'd have to move to a bigger office, hire more help, and work sixteen hours a day. The hospitals would court me, the pharmacists, cardiologists, and chest surgeons would love me, and I wouldn't have to pay that practice-building outfit thousands of dollars for their secrets.

'Course I guess my ideas might be considered a little unethical by some folk. But I'm only a doctor, not a Madison Avenue city slicker. Maybe marketplace ethics aren't the same as medical ethics.

For all I know, medical ethics are as old-fashioned and obsolete as I am.

*—MRJ*

# The Right Thing for the Right Reason

As the legislatures of some states pass oppressive legislation requiring physicians to accept mandatory assignment, I see no signs on the horizon that similar legislation will be passed requiring the gas company, the electric company, or grocery stores to accept 50% on the dollar as payment in full for their goods and services.

While this must be dealt with in a different way and at a different time, there is a significant group in our patient population that needs our help now. This nothing new. Physicians have always honored our commitment to mankind and cared for this group of people without fanfare or accolades.

It has always been a problem, however, to correctly identify some of these individuals without means testing or other screening devices.

Tulsa County Medical Society is to be congratulated for their solution to this problem. Under the leadership of Dr Rollie Rhodes and Dr George Prothro, and with the cooperation of some senior citizens groups, they have devised a unique method to identify these truly needy individuals — they call it the VIP (Very Important Patient) Program.

The House of Delegates of the OSMA rightly recognized the potential of this program and has directed your leadership and staff to assist the various county societies throughout the state in implementing similar programs.

With the economy of our state in its present severely depressed state, there are increasing numbers of persons in all population groups who are in need of medical care.

While this is in reality a social problem which should be shared by all of society, physicians and organized medicine have always been in the forefront of solutions to these problems.

I have asked Dr Prothro and Dr Rhodes, leaders and developers of the Tulsa County program, to head up a task force to deal with this problem. (They know where the rocks are.) However, this is a physician-community problem and it will take the cooperation of all of us to make it work in a fair and efficient manner.

I ask each of you to cooperate with the OSMA, your county medical society, and the task force in helping solve this important problem.

In my opinion, this program is one of the highest priorities of our association at this time.

Doing the right thing for the right reason is important.

M. Joe Crosthwait, MD

# Outpatient Multidisciplinary Geriatric Assessment I

(First of two parts)

JAMES W. MOLD, MD; JEFFREY R. STEINBAUER, MD; SHIRLEY C. WUNDER, RN; BEVERLEY SMALL, RN

**Optimal assessment of complex geriatric patients is qualitatively and quantitatively different from traditional medical evaluation. Important principles include teamwork and an emphasis on function.**

Outpatient multidisciplinary assessment of geriatric patients involves a team approach to the evaluation of complex biomedical, functional, psychosocial, and environmental problems that seems to be useful in planning therapy for this group of patients. Although this technique is costly and time-consuming, the benefits in terms of improved outcomes and total health care costs appear to justify the initial investment of time and money. The Geriatric Multidisciplinary Evaluation Clinic (GMEC), operating out of the O'Donoghue Rehabilitation Institute at the University of Oklahoma Health Sciences Center, has been in operation for over a year. Information regarding the first 68 patients seen and evaluated in that clinic is presented, two representative cases are described, and certain issues regarding the operation of a geriatric assessment clinic are discussed.

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## Overview of Geriatric Assessment

The term *geriatrics* has traditionally been used to refer to the medical care of older patients, that is, those over the age of sixty-five years. Increasingly, however, it is coming to mean primarily the care of a particular subset of older people also referred to as the "frail" elderly. These are physiologically aged individuals with multiple biomedical, functional, and psychosocial problems. Their medical care requires certain specialized knowledge and skills, and a somewhat different general approach from that used in the care of other patients. The "subspecialty" field of geriatrics might then be compared to neonatology at the other end of the age spectrum.

An essential component of good medical care is a complete assessment of the patient and his problems, since without an accurate assessment, the most effective treatment plan cannot be developed. The assessment of frail geriatric patients is, of necessity, qualitatively and quantitatively somewhat different from the assessment of other patients. It should include, at minimum, a biomedical evaluation, a functional evaluation, a psychosocial evaluation, and an inventory of available resources. Additional valuable information can frequently be obtained from a home visit and a family interview.

Biomedical problems are usually multiple and complex and many are unique to this group of

patients. Most physicians have had very little specific training in the evaluation of problems such as recurrent falls, urinary incontinence, dementia, drop attacks, and osteoporosis, all of which are extremely common geriatric disorders. Many conditions that have previously been attributed to normal aging, such as osteoarthritis, presbycusis, macular degeneration, systolic hypertension, and memory loss, are now felt to represent abnormalities that deserve more careful investigation and/or treatment.

**Geriatric patients tend to have one or more functional disorders.**

Geriatric patients tend to have one or more functional disabilities which interfere with their enjoyment of life. Amelioration of these functional impairments is often more important for the health and well-being of these patients than treatment of their biomedical problems. Therefore, a functional assessment is an essential part of the evaluation. A patient's ability to function may not correlate at all well with the severity, total number of, or adequacy of management of his/her biomedical problems. Specific measures can often be taken to improve a patient's level of functioning that are unrelated to the management of the individual biomedical conditions. For instance, raising the height of the chairs in the house of a patient with osteoarthritis may decrease knee and back pain previously aggravated by the difficulty associated with getting in and out of the standard lower chairs, thereby increasing the patient's ability to ambulate.

Psychological problems resulting from, or aggravated by, functional decline and other losses and by biomedical problems and their treatment are common in geriatric patients. Therefore, comprehensive assessments of social and family support, mood, use of alcohol and other addictive substances, and mental status are imperative. In many cases, it is important to determine the patient's competency to make decisions regarding his or her own care. Depression, grief, suicidal ideation, alcoholism, social isolation, dementia, abuse, and neglect are common geriatric psychosocial problems, which are not always easily recognized.

To help address these multifaceted problems, a complex array of resources now exists in most communities. Many resources have been developed specifically to help frail elderly people maintain their independence for as long as possible. Problems such as housing, transportation, meal preparation, home maintenance, companionship, etc, can often be solved through proper use of available resources. Many frail elderly patients have significant financial problems and may not be aware of some of the benefits to which they are entitled. For these reasons, it is necessary to assess each patient's financial situation and the need for and ability to qualify for available services. While adequate resources may exist, it is nearly impossible for primary care physicians to know about all of them. Mobilizing these resources also takes time that busy physicians often can't spare.

Frequently, nursing home placement becomes a consideration for many patients and their families. This is a major decision that too often is made without a full investigation of alternatives. Many people are placed in nursing homes inappropriately because of inadequate preadmission assessments and lack of knowledge of treatment options and community resources.

Because of the complexity of problems encountered in these different spheres, there is no other branch of medicine in which an interdisciplinary team approach to assessment and treatment is more important than in geriatrics. In fact, this approach is quickly becoming standard procedure on geriatric inpatient units, consultation services, in nursing homes, and in geriatric assessment clinics. Geriatric assessment teams commonly include a physician, a social worker, and a nurse, nurse practitioner, or physician's associate. In addition, some teams include a psychologist, a physical therapist, an occupational therapist, and/or a dietician or nutritionist. Specialists in other areas are included as needed in particular cases.

**Psychological problems are common in geriatric patients.**

Information regarding the benefits of multidisciplinary geriatric assessment, as well as when and to whom it should be applied, is gradually acccumulating. Rubenstein reviewed the evidence from published studies prior to 1983, most of which were descriptive, quasi-experimental, or uncontrolled. He concluded that comprehensive geriatric assessment probably results in improved diagnostic accuracy, priate placement decisions, improved functional status following treatment, more appropriate use of community resources, and more appropriate use of medications.[1]

**Patients are evaluated only — not treated.**

Rubenstein later published the results of his own randomized prospective trial of geriatric assessment and treatment on a special geriatric inpatient unit in a VA hospital.[2] In a rather carefully selected group of frail elderly inpatients randomly assigned to either standard care or the geriatric assessment unit (GAU), he was able to demonstrate that evaluation and treatment in the GAU resulted in substantial improvements in mortality (23.8% vs 48.3%), placement back in the home (73% vs 53.3%), number of rehospitalizations (34.9% vs 50%), and overall cost of care over the subsequent year. The patients that were included in the study were at least 65 years of age and had persistent biomedical, functional, or psychosocial problems that interfered with discharge to their homes. Patients were excluded if they had severe irreversible dementia or other irremediable and disabling conditions (multiple sclerosis, end stage cirrhosis, etc), could not perform more than three of six activities of daily living, had insufficient social or family supports to make return home a reasonable possibility, or were functioning well and would definitely return to the community without any special help.

In the October 1985 issue of the *Journal of the Oklahoma State Medical Association*, Mohr reported the results of inpatient geriatric assessment and treatment at the O'Donoghue Rehabilitation Institute (ORI) from 1981 through 1983.[3] Patients admitted to this unit were felt to be at high risk for institutionalization, but only 7.5% required nursing home placement at the time of discharge. Geriatric inpatient services continue at ORI through both the Department of Internal Medicine and the Department of Family Medicine.

One of the earliest *outpatient* geriatric assessment clinics in the US was started in Rochester, New York, in 1970 as a result of the "Health Care of the Aged" study conducted by the Genesee Region Health Planning Council, which found, among other things, that well over half of the patients in long-term care institutions in the Genesee region were receiving inappropriate levels of care. It was recommended that "a special service be established for diagnosis, evaluation, and placement of chronically ill and elderly persons." Williams (T.F.) summarized the methods and results of the first 30 months of operation of that clinic, which was established in the outpatient department of a rehabilitation/geriatric hospital affiliated with the University of Rochester.[4] This clinic was very successful with regard to its original mission and continues to serve this and many other functions. Williams (M.E.) and Williams (T.F.) in 1986 described the current operations of the clinic and discussed the process of outpatient geriatric assessment in general.[5] The Duke University Older American Resource Services/Geriatric Evaluation and Treatment Clinic (OARS/GETC) is another example of a very successful multidisciplinary geriatric evaluation clinic which has been in operation since 1978.[6]

In a randomized controlled trial of multidisciplinary team assessment of geriatric patients versus assessment by private internists, Williams (M.E.) demonstrated that multidisciplinary assessment of frail elderly patients resulted in a significant reduction in health care expenditures over the subsequent year. The average health care cost per person per year in the group that had a team evaluation was approximately $2000 (25%) less than for the internist-evaluated group (unpublished data).

**All recommendations are sent to the private physician for consideration and implementation.**

This was due primarily to fewer days spent in the hospital. Health care costs included hospitalization, nursing home, home health care services, and physician visits. The internists were paid whatever they felt their evaluation was worth. They were felt to be among the most clinically competent physicians in the Rochester area, and many had had some specific geriatric training.

## The Geriatric Multidisciplinary Evaluation Clinic

Based upon the reports from Rochester, the author's (J.M.) experience with the Geriatric Evaluation and Treatment Clinic (OARS/GETC) at Duke University, and an intriguing article by Campbell and Whitenack regarding the evaluation and treatment of "multiproblem patients,"[7] the Geriatric Multidisciplinary Evaluation Clinic (GMEC) was created in May 1985 at the O'Donoghue Rehabilitation Institute. The philosophy and structure of the GMEC, a summary of the first 68 patients seen, some case examples, and some of the problems/issues that remain to be solved are the subjects of the remainder of this article.

Although geriatric evaluation clinics should theoretically be quite helpful to private physicians who would find it hard to mobilize the other important members of the team and spend the necessary time assessing these patients in the office, the experience of most geriatric evaluation clinics has been that only a small percentage of referrals come from physicians. GMEC was specifically designed to try to overcome some of the barriers to physician referral. Patients are evaluated only — not treated — and all recommendations are sent directly to the private physician for consideration and implementation. With the exception of certain social service referrals, no referrals are made, no medications are changed, and no tests are ordered if the patient has an ongoing relationship with a primary physician. This differs significantly from the majority of geriatric clinics described in the literature. Every effort is made to return the patient promptly to his/her primary physician. Patients without physicians are referred to physicians in their area who have been identified as being interested in the care of geriatric patients. In the vast majority of cases, the whole evaluation is done in one afternoon (3 to 4 hours). One return visit is scheduled a month later, primarily to be sure that follow-up medical care has been obtained and that the GMEC report was received and considered, to assess the effectiveness of the team's suggestions, and to make new or alternative suggestions if needed.

The core evaluation team is made up of a nurse/coordinator, a physician, a social worker, a physical therapist, and an occupational therapist. The clinic staff meets weekly in the outpatient department of the O'Donoghue Rehabilitation Institute. After receiving a referral, a nurse conducts a screening interview by telephone with the patient or a family member or care giver prior to the clinic visit. The information obtained in this interview covers demographic data; social, dietary, and some medical history; and environmental safety issues. A family genogram is constructed. Patients are asked to bring all over-the-counter and prescription medications with them to the clinic. Pertinent medical records are requested and decisions are made regarding the need for input from other specialists such as a psychologist, psychiatrist, physiatrist, speech therapist, or nutritionist in addition to the core team.

When the patient arrives at the GME clinic, a nurse gathers more general information, including medication and diet histories and typical daily activities. Vital signs are obtained, including pulse and blood pressure taken in three positions. The nurse then administers the Folstein Mini Mental State Inventory, a cognitive assessment,[8] and, when indicated, the Elderly Mood Assessment Scale, a screen for depression.[9] Hearing and vision are evaluated. Family members are interviewed, and family interaction patterns are observed.

An occupational therapist and a physical therapist work together to evaluate the patient's balance, strength, mobility, and ability to perform activities of daily living. Safety issues, as they relate to independent living skills, are also assessed. The social service evaluation includes an inventory of social and financial needs as well as knowledge of and access to community services such as day care, congregate meals, housekeeping, shopping services,

**The goal is to maximize quality of life and independence for the patient.**

etc. The physician takes a complete medical history and performs a physical examination.

The entire team meets when the individual assessments have been completed. Team members discuss the patient's needs and available resources as identified by the patient, the patient's family, and the evaluation team. A prioritized problem list is constructed and possible solutions are discussed. The goal is to maximize quality of life and independence for the patient for as long as possible.

The team physician then sends a letter to the patient's physician and to the patient/family/care giver which includes the prioritized list of identified problems and suggestions for their management. Two weeks following the visit, a postcard is mailed to the patient's physician to determine whether the patient has returned for discussion and implementation of the suggestions and to obtain feedback regarding the perceived value of the GMEC report.

Because of the time involved in team assessment, a maximum of only three to four patients can be seen during a single clinic session. Late cancellations, rescheduling, and no shows have been a significant problem at times. This problem has been partially solved by scheduling one extra patient from the following week's clinic as an alternate.

The clinic's services and physician's fees are reimbursable by Medicare, which covers 80% of the charges. Supplemental insurance often covers the remaining charges. O'Donoghue Rehabilitation Institute files Medicare and insurance claims for patients.

**Note:** The second part of this paper will appear in next month's JOURNAL.

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## Coming in July . . .

Being prepared for publication in July is Part II of "Outpatient Multidisciplinary Geriatric Assessment." Several other papers are also in production, and of course, the issue will feature the complete proceedings of this year's OSMA Annual Meeting in Oklahoma City.

# Love vs Attachment

JOHN E. POARCH, MD

**As physicians, we see multitudinous problems directly or indirectly related to lack of discipline. Overstimulation and overprotectiveness are impediments to growth and development. The following article attempts to clarify the essential role of limits (discipline) in dealing with both impediments, thus fostering maturation.**

Love is a feeling. Attachment is a psychological bond. Proof for their separateness is as close as family settings.

Some families show attachment by investing the family name with immense pride, and the bond between members is obviously very strong, though they often seem to lack the ability to *feel* close, warm, loving, or genuinely intimate. Love, as a feeling, is inhibited, almost as though the ability to express or receive affection is inversely related to the strength of the family bonds.

In some families, close feelings are so threatening that even if family members come together only on rare occasions, such as weddings or funerals, there nearly always ensues one or more intense quarrels.

Since I come from a family much like, but not quite to the extreme I described above, this phenomenon has been more than just a curiosity or a passing interest. It has only been of late that I have arrived at what I currently feel is some possible understanding, which I attempt to clarify in this communication.

I view love as a feeling that comes and goes, just like any other feeling. One would not expect to feel angry or sad all the time, but it seems that most people assume that feelings of love should be constant, and that something is wrong if they are not. I consider attachment as a psychological tie between people that reflects their relative degree of fear of losing the other person, or fear of being alone.

A common assumption is that love and attachment are the same thing. I have discovered that it's very common for the bonding or attachment between family members to be very strong and the ability to feel close or feel warm and intimate, to be relatively limited.

Two concepts are important to understand prior to continuing with the central theme of the difference between love and attachment. Their effects illustrate my point.

The first is overprotectiveness. The overprotective person feels responsible for, and has the need to take care of everyone, as though people could not take care of themselves. This necessarily includes giving relief if anyone has physical or emotional discomfort. The overprotective individual takes great pride in this attitude, and feels it is what makes him or her a good person, thus gaining a sense of value or self-worth.

(In my opinion, just as is a kitten or a puppy unique, we are as precious and special as we can ever be the day we are born. The reason we don't feel value later is because of the defenses we build up. As we grow up we become overly excited by experiencing

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fully the special infant within us and nervous to be aware of its presence consciously.)

Overprotective people hear only one message in complaints from their children, spouses, or friends: "You are not a good person (or spouse, or parent), or else you would keep me comfortable like a baby, and I would not have any complaints or criticisms." Even such statements from a spouse as, "You are not treating me like an equal," or, "You are not viewing me as being able to be responsible for myself or capable of taking care of myself," are only heard as "You are bad."

Overprotective people are often so compliant, or helpful, that they provoke everyone around them. It is not uncommon for them to become very helpful and accommodating in order to try to achieve a desired closeness while not comprehending that this very helpfulness ends up emotionally distancing (because it is insulting) the person desired. I say insulting because it treats the other person as a weaker being.

A complicating deception in overprotectiveness is that the typical behavior to achieve closeness is simultaneously an unconscious subterfuge to prevent it. Overprotective people cannot tolerate intimacy. They initiate behavior and attitudes that are intended to gain merit for intimacy while protecting them against closeness. Changed attitudes and behavior in this reaction pattern are very difficult to attain because the overprotective person must give up something that makes him or her feel very worthwhile.

Sex is an obvious manifestation of closeness. Many patients complain of having no sexual desire for their mates. More frequently than not, the underlying reason is that they are not being honest with one another, in order to avoid conflict. They are being overprotective. The woman who pretends in order not to "hurt his ego" is insulting her husband. The man who performs or gives when he doesn't genuinely want to do so is treating his wife as though she is weak and threatening simultaneously. He thinks he does it because he cares, but he does it because he is afraid of her disappointment and anger.

The second concept to clarify is incestuous anxiety, that is, incestuous excitement leading to anxiety. Your most likely response to these words will be disgust, since disgust is a relatively universal defense against incestuous wishes. Important to understand is that incestuous desires and impulses, as used here, are not meant to convey any negative connotation. All our sexual and romantic feelings originate toward family members. They are as natural as anything can be.

**We are as precious and special as we can ever be the day we are born.**

By incestuous anxiety I refer to that nervousness that occurs when sexual thoughts or feelings about siblings, parents, or children come closer, or actually into, awareness. This explains why you may have responded to the last paragraph with denial. You perhaps said, "Not me," which would be a psychological defense to keep the ideas from conscious awareness. Your reaction is to my tension-producing statement. Less conspicuous manifestations, that might not be threatening, would include a mother's jealousy of her son's girlfriend or wife, and a father's

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| Intimacy<br>Maturity<br>Emotional growth<br>Limits — Firm yes/no<br>**LOVE** | **vs** | **ATTACHMENT**<br>Overprotectiveness — Lack of limits<br>Overstimulation<br>Immaturity<br>Behavioral and emotional problems |

preoccupation with the fear that his daughter will be raped.

Children give us obvious examples. It is common for a child about three years old to express the desire and intention to marry the parent or an older sibling of the opposite sex. Most of the fairy tales that we love so much at age four or five were written about our earliest incestuous romantic feelings. Hence, the witch or mean stepmother is the adversary for girls, and the dragon or monster is the adversary for boys.

**Overprotective people are often so compliant that they provoke everyone around them.**

As we grow, we learn that these desires cannot come true, and they are gradually transferred to people outside the family. In the process, usually as early teenagers, it is common to have a secret crush on an adult outside the family, such as a teacher, minister, or coach.

The impossible dream, the Oedipal wish come true, is, in families with strong bonding, as scary as death or craziness. It is quite common for five-year-old girls to wake up in terror, complaining of witches or spiders, and five-year-old boys to wake up crying, talking of gorillas and monsters. Such dreams are the rule rather than the exception in our culture. In teenagers, mythical fears about masturbation are related to this fear of incest. Self-stimulation carries ominous punishment. Teenagers hear, "You will go blind," or, "It will make you go insane."

Love and attachment, as feelings and psychological ties, show the effects of protectiveness and resulting incestuous anxiety. Roderic Gorney, MD, in an award-winning paper in the *American Journal of Psychiatry,* October 1971, pointed out the immensely important variables that seem to correlate almost directly with the intensity of the mother/child attachment, which later determines the family attachments. The variables include competitiveness arising out of the desire to keep mother all to oneself, that makes us all want to be number one at everything we do, and resulting achievement orientation. This necessarily leads to progress in the culture, but also predisposes tendencies toward violence and emotional problems. In cultures and families where mother/child attachment is minimal, such as in some Tahitian tribes and in the early kibbutz system, all the variables mentioned seem relatively lower and, in addition, Oedipal or incestuous anxiety is relatively minimal.

Sex is biologically the goal in relating and *ideally,* in humans, the ultimate positive expression of feeling between two people. Because of this, when a parent gives an unsure message about anything, such as, "Well, maybe so," or, "I don't know, I'll think about it," it implies to the child that they can have more. The child doesn't know where the line is drawn. Children will, therefore, continue to press in order to get a line or limit. The lack of one implies that the ultimate goal, sex, could possibly come true. Obviously, this is overstimulation. Whining, complaining children will continue to do so until a definite clear "No" is provided for their behavior.

This effect of overstimulation depends on our level of tolerance to stimuli, which is conditioned during childhood. More or continued stimuli above that level results in overstimulation or incestuous anxiety. I use these two terms interchangeably because *all positive feelings push the same button in the brain,* the pleasure center. It makes no difference whether the stimulation is freedom, achievement, excitement, money, prestige, power, or intimacy. Where the thermostat is set depends on the tolerance of the people around us when we are very small. An example would be how a mother responds when her child gets muddy, or is playing with feces in the bathtub. The person with little tolerance might scream, and the one with great tolerance might say warmly, "You little rascal, you."

The degree of incestuous anxiety increases in direct proportion to the strength of attachment. The ability to *feel* love and closeness and to tolerate intimacy (this includes sexual feelings) depends on a person's ability to give a clear yes or no even at the risk of losing the relationship, which is experienced as losing love. An example would be a parent's reluctance to discipline a child, or say no to a spouse, for fear of losing the person.

There are many, many ways people lessen the stimulation, and thus reduce the anxiety. They press behaviorally for discipline by destroying property. Children break toys, teens and adults break speed limits, ignore time restrictions, and forget agreements and appointments. They progressively become more offensive emotionally, showing hostility, sullenness, or withdrawal. They may block off

feelings, such as intimacy and love, from consciousness to defend against the threat of closeness. Overeating, accidents, and illness are also ways of reducing stimulation when it is far too much to be tolerated. Is it any wonder that many children are more unruly, daydream more, and function much less effectively prior to birthdays and Christmas? Holiday behavior is a temporary example of the overstimulated person's life-style.

Ultimately, the more one has ability to give an unmistakable yes or no and provide definite consequences for transgressions, with limits set in response to the child or another person's need, rather than from fear and overprotectiveness, the more likely one can *feel* loving and intimate.

**Limits reduce incestuous anxiety, thereby freeing the emotions.**

Limits reduce incestuous anxiety, thereby freeing the emotions. Discipline out of overprotectiveness would include examples like, "Put your coat on. It's cold outside." And, "Don't climb the tree. You will fall." Or, "I don't want you to see that boy (or girl) again."

An example of discipline in response to a youngster's need would be taking away Saturday morning cartoons as a consequence for not remaining in bed after bedtime. This might, surprisingly, result in a child who is pleasant and agreeable on Saturday, as opposed to complaining and unpleasant to be with on Friday. Grounding a teenager for one week after coming in late may result in the youngster's being again pleasant to live with, as opposed to the sullen, disagreeable, complaining person he or she was prior to that evening's outing. Another example would be taking driving privileges away from a teenager after a speeding ticket is received, resulting in less time spent withdrawing in the bedroom, a greater tendency to talk to parents and, in general, more effective functioning at home and at school.

The statement I most often use in my office to drive home the importance of the issue is, "A son's ability to say no to his own sexual desires and impulses and to his girlfriend's sexual desires and impulses is in direct proportion to his mother's ability to say no to him regarding matters unrelated to sex." Obviously, the same is true between fathers and daughters. Often the father that is most concerned about his daughter's getting pregnant or being taken advantage of is the one who is most protective and indulgent and least able to say no to her.

Families with intense bonding, that have the most trouble tolerating any genuine warm feelings because of unconscious heightened incestuous anxiety, such as the type described earlier, experience close feelings and are aware of love only if they live great distances apart. The farther apart they are, even on opposite sides of the world, the better. They would, of course, be more able to experience close feelings safely in times of great emergency, such as in case of serious injury, illness, or death in the family.

In summary, the benefits of honest, clear, unequivocal limits, with unavoidable consequences for exceeding them, are these: maturity, increased likelihood of achieving one's full potential, and increased tolerance for feelings of all kinds, including intimacy. In addition, there is the bonus of decreased likelihood of emotional disorder. If we can dare to risk loss of our attachments, we not only preserve our relationships, but we also increase the possibility for fulfillment of love as more than a wish, an ideal, or just lip service. Love can become a genuinely intense emotion that orders life and disentangles attachments.

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News from
the Oklahoma State
Department of Health

## High Blood Pressure

In recent years, cardiovascular and heart disease mortality has decreased. Clinical studies have demonstrated the benefits of reducing blood pressure as one of the mechanisms. While these benefits have been achieved primarily by pharmacologic agents, the 1984 *Report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure* suggested that the benefits of these agents must be balanced against the potential for long term adverse effects by drug treatment. At moderate and severe elevations of blood pressure, benefits outweigh risks; but at milder levels (90-104 mmHg diastolic), these benefits may not be compelling.

Nonpharmacologic therapy provides options for high blood pressure management not only for those patients with milder levels of high blood pressure, but as adjunctive therapy for those with higher pressures who require drug therapy. *Nonpharmacologic Approaches to the Control of High Blood Pressure* is a report which reviews three approaches to control high blood pressure. The three approaches reviewed are modalities of weight reduction, control of alcohol intake, and restriction of dietary sodium.

For a copy of *Nonpharmacologic Approaches to the Control of High Blood Pressure,* contact the Chronic Disease Control and Eldercare Division, Oklahoma State Health Department, (405) 271-4072.

Another report available through the division is the *Statement on Hypertension in the Elderly* developed by the National Institutes of Health. This report addresses issues related to prevalence, guidelines on management, including antihypertensive drugs of choice and their dosage, goal pressure for people over age 65, and side effects specific to older people.

The report serves two purposes: (1) to assist physicians in their care of the older hypertensive patient, and (2) to guide health professionals participating in high blood pressure control programs that serve the elderly population.

| DISEASE | March 1987 | TOTAL TO DATE | | |
|---|---|---|---|---|
| | | This Year | Last Year | 5 Yr. Avg. |
| AMEBIASIS | 0 | 2 | 1 | 4 |
| CAMPYLOBACTER INFECTIONS | 14 | 43 | 39 | — |
| ENCEPHALITIS, INFECTIOUS | 4 | 7 | 4 | 6 |
| GIARDIA INFECTIONS | 16 | 51 | 42 | — |
| GONORRHEA (Use ODH Form 228) | 744 | 2423 | 3030 | 3664 |
| HAEMOPHILUS INFLUENZAE INVASIVE DISEASE | 14 | 36 | 59 | — |
| HEPATITIS A | 30 | 85 | 77 | 129 |
| HEPATITIS B | 17 | 58 | 34 | 54 |
| HEPATITIS, NON-A NON-B | 3 | 12 | 12 | — |
| HEPATITIS UNSPECIFIED | 3 | 16 | 14 | 42 |
| MEASLES (RUBEOLA) | 0 | 1 | 1 | 0 |
| MENINGITIS, ASEPTIC | 4 | 12 | 11 | 13 |
| MENINGITIS, BACTERIAL (non-meningococcal, non H. Influenzae) | 5 | 16 | 19 | 19 |
| MENINGOCOCCAL INFECTIONS | 2 | 11 | 9 | 11 |
| PERTUSSIS | 16 | 29 | 20 | 20 |
| RABIES (Animal) | 2 | 5 | 14 | 34 |
| ROCKY MOUNTAIN SPOTTED FEVER | 1 | 1 | 1 | 2 |
| RUBELLA | 0 | 0 | 0 | 0 |
| SALMONELLA INFECTIONS | 20 | 49 | 50 | 70 |
| SHIGELLA INFECTIONS | 32 | 61 | 28 | 59 |
| SYPHILIS (Use ODH Form 228) | 16 | 41 | 45 | 55 |
| TETANUS | 0 | 0 | 0 | 0 |
| TUBERCULOSIS | 31 | 56 | 48 | 71 |
| TULAREMIA | 2 | 5 | 1 | 1 |
| TYPHOID FEVER | 0 | 1 | 1 | 0 |

| Diseases of Low Frequency | Total to Date This Year |
|---|---|
| ACQUIRED IMMUNE DEFICIENCY SYNDROME | 17 |
| BRUCELLOSIS | 0 |
| LEGIONNAIRES DISEASE | 4 |
| MALARIA | 2 |
| REYE SYNDROME | 1 |
| TOXIC SHOCK SYNDROME | 7 |

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*Dr Lin Yen-Tchen*

# Chinese physician visits state, builds another bridge

Readers of the JOURNAL may recall an illustrated article in the January 1986 issue — "Bridge to Understanding: The China Connection" — about the experience of a group of physicians who visited hospitals in China in the fall of 1985. They were members of the Los Angeles Chapter of Physicians for Social Responsibility and other PSR doctors from around the country. The purpose of their visit was to discuss the prevention of nuclear war with their Chinese colleagues. The group also visited Hiroshima and were in Tokyo when International Physicians for the Prevention of Nuclear War was awarded the 1985 Nobel Peace Prize.

While in Shanghai, touring the Rui Jin Hospital, the teaching facility of Shanghai Second Medical University, their host, Dr Lin Yen-Tchen, chairman of the department of surgery, expressed the desire to visit the United States again. He had been here in 1979 as guest of the American Friends Service Committee of Philadelphia.

Through the effort of Robert C. Hardy and Robert G. Hirschi, DDS, the financial support of the OU Foundation, Dr Kenneth Fong and the PSR Chapters in St Louis and Oklahoma City, and the hospitality of Dr G. Rainey Williams, chairman of the department of surgery at the OU College of Medicine, Dr Lin is currently enjoying a four-month sojourn through the United States to observe the current state of the surgical art as it is practiced in America. When he completed his four-week stay in Oklahoma, during which he attended a meeting of the American Surgical Association in West Palm Beach, Florida, courtesy of the College of Medicine, he said his April experience in Oklahoma had been highly rewarding and thought the remainder of his trip would be equally beneficial.

During May, June, and July, Dr Lin is being hosted by some of the people who visited China and by the chairmen of surgery in other universities. His rather extensive itinerary includes experiences at:

University of North Carolina — Dr Colin Thomas, Chief of General Surgery, UNC Memorial; Dr Gerald A. Drake, PSR

Harvard University — Dr William Silen, Chief of Surgery, Beth Israel Hospital

Washington University, St Louis — Dr Samuel Wells, Chairman of Surgery; Dr Kathleen Garcia, PSR

University of Wisconsin, Milwaukee — Dr Robert Condon, Chairman

*(continued)*

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## ACP mastership awarded to internist C. S. Lewis, Jr., MD

C. S. Lewis, Jr., MD, Tulsa, was recently awarded mastership in the American College of Physicians (ACP). The honor was announced at the 68th annual meeting of the ACP in New Orleans.

Dr Lewis was one of only 13 medical educators and investigators to be selected for mastership this year. Masters of the ACP are honored for personal character, eminence in practice or medical research, achievements in medicine or science, and service to the ACP; they are authorized to use the letters MACP after their names in recognition of their accomplishments.

A private practitioner in internal medicine, Dr Lewis was honored for his service to the ACP. He was the 1986-87 president of the college and served as ACP governor for Oklahoma from 1975 to 1979. Prior to that he was chairman of the ACP Board of Governors. From 1983 to 1985 he served as ACP treasurer, and in 1986 he completed six years as an executive committee member of the ACP Board of Regents.

Dr Lewis, a 1945 graduate of Washington University School of Medicine in St Louis, was 1977-78 president of the Oklahoma State Medical Association. He is a staff physician at St John Medical Center, where he has served as chief of staff (1963), chairman of the Department of Medicine (1970-71), medical director (1976-78), and a member of the Board of Directors (1976-84).

## Chinese physician (continued)

University of Nebraska — Dr Byers W. Shaw, Jr., Chairman

UCLA and/or USC — Dr Samuel I. Roth, PSR; Dr Richard G. Saxon, PSR

University of California, San Francisco — Dr Rudi Schmidt, Dean, College of Medicine

Dr Lin will depart for Shanghai on July 31st.

As Dr Lin previously explained, modern Chinese medicine needs state-of-the-art diagnostic and treatment technology as well as means to expand basic science and clinical research efforts.

Dr Williams firmly believes this small effort involving person-to-person diplomacy affords one more "bridge to understanding" between medical practitioners around the world.

*Walls elected president*

# Tulsa hosts annual state convention for family physicians

The Oklahoma Academy of Family Physicians (OAFP) held its annual meeting April 8-11 in Tulsa at the Sheraton Kensington Hotel. Over 250 family practice physicians attended the four-day event.

The Oklahoma Academy of Family Physicians was organized in 1947 and has a statewide membership of approximately 1,000 family doctors. During the 1987 annual assembly, the new officers were installed. Elected president for 1987 was Les Walls, MD, associate dean and chairman of the family practice department at the University of Oklahoma Tulsa Medical College.

The national executive vice president of the American Academy of Family Physicians (AAFP), Robert Graham, MD, was a guest speaker during the assembly of family physicians.

In addition to a variety of workshops led by members of the academy, many pharmaceutical companies had technical exhibits on display during the convention. Additionally, Mayor Dick Crawford was the featured speaker at the business luncheon on April 9 and presented a proclamation designating April 6-10 as Family Physician Week in Tulsa.

"The academy meeting is a scientific and business session designed to provide continuing education for family doctors," said President Les Walls, MD. "It is important to continue learning because it ensures high quality medical care for our patients."

The Tulsa economy also benefitted from the meeting. The Oklahoma Academy of Family Physicians brought an estimated $125,000 in revenues to the city of Tulsa during their four-day meeting, said Ken Bridenbach, director of the convention and special events division of the Tulsa Chamber of Commerce.

When the physicians were not busy with workshops, they participated in such activities as a photography contest and an antique auction.

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# SMOKING...

# ... a habit that *dies hard*™

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*Just for the health of it*

## Reader contributes his own interpretation of smoking

The JOURNAL of the Oklahoma State Medical Association has always encouraged and sought original contributions from its readers. Of special note recently was a submission from Steven W. Newell, MD, an ophthalmologist in Enid, whose important message filled but a single page.

In his cover letter, Dr Newell explained that he had once heard one of his patients describe something as "dying hard." The vivid phrase stuck with him and sparked an idea for an anti-smoking statement.

Dr Newell took his idea to a local high school art teacher, Jim Giles, and the result was the disquieting illustration "Smoking . . . a habit that dies hard," seen on page 382.

The drawing has provoked favorable comments from several of Dr Newell's patients and also from physicians. Both he and the JOURNAL hope that its publication will be of interest and benefit to physicians and patients across the state.

## ACC governor for state is Tulsa's R. W. Neal, MD

R. W. Neal, MD, FACC, Tulsa, has been elected College Governor for the State of Oklahoma by the American College of Cardiology's (ACC) Board of Trustees.

Dr Neal is currently in private practice in Tulsa and is medical director of the cardiac catheterization laboratory at St John Medical Center. He is clinical associate professor of medicine at both the University of Oklahoma Tulsa Medical College and the University of Oklahoma Health Sciences Center, Oklahoma City. He was graduated from Phillips University, Enid, and received his MD degree from Washington University School of Medicine, St Louis.

College governors, who serve three-year terms, are responsible for reviewing all applications for membership to the college within their areas and for making recommendations to the college's credentials committee. The college governor is the official representative of the college within his or her area and assists in monitoring legislation and regulations relevant to cardiovascular medicine at the state level.

## Lipid research in OKC to include fat absorption in infant feeding

A cooperative study on infant feeding will be conducted by Baptist Medical Center and the Oklahoma Medical Research Foundation as part of a continuing study on how lipids (fats) are carried in the blood during infancy and childhood. The announcement was made by Daniel Lane, MD, PhD, and Walter McConathy, PhD, Oklahoma City, co-investigators on the project.

"Our earlier studies have been on infants during the first month of life and most recently have emphasized differences in the kind of fat in formula. This study will use a formula with a fat content like breast milk to determine if the fat is absorbed the same as that of breast milk. We will also see if supplementing breast milk with the same formula changes how the fat is absorbed," said Dr Lane.

Three groups of infants will be recruited from Baptist Medical Center prenatal classes and followed until solid foods are added to their diets. Both growth and blood lipids will be evaluated during the study, and no side effects are expected. The project is expected to last one year and is funded in part by Wyeth Laboratories, Philadelphia, Pa.

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# Bethany physician knows you've come a long way, Babies

By now most physicians in Oklahoma probably know that one of their own succeeded last fall in his bid for a seat in the Oklahoma legislature. What many of them may not know is just how he did it.

John W. Bumpus, MD, Bethany, now a member of Oklahoma's House of Representatives, was not a seasoned politician when he began his run for office; he was a concerned physician who wanted to get involved. And he wanted the voters to know that.

How could he let them know he was a doctor without making it the focal point of his campaign? Simple. The "Bumpus Babies."

Dr Bumpus's campaign workers, the Bumpus Babies, were precisely that — some 250 energetic young people, all delivered by Dr Bumpus during his 26 years of practice.

"It was the perfect way of telling people 'I'm a doctor' without campaigning as an MD," says Robert Baker III, director of the Oklahoma Medical Political Action Committee (OMPAC), "as well as showing the voters that the Bumpus campaign had a very diverse group of supporters."

The campaign got quite a boost from the state's medical community, starting with campaign manager Alice (Mrs Kenneth) Whittington, an OSMA auxilian and wife of a family practitioner. From a crowded field of candidates, Dr Bumpus ultimately emerged as the front-runner, winning the open seat on November 4th with almost 70% of the vote.

Dr Bumpus is the first physician to win a seat in the Oklahoma legislature in many years and is the only one currently serving.

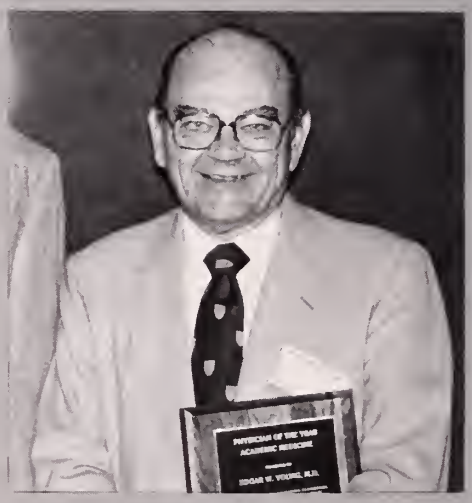
# Don't cry. Just wait.

Don't cry at my funeral.

I dare you to believe, as I do, that what happens is God's will. I haven't always understood this will and I haven't always followed what I thought His will would be. But, I, like all mortals, have always been weak. When I have been the strongest have been those times where I saw most clearly what He wanted me to do.

I believe in God and have felt His love, although I have not behaved in a conventional way to demonstrate that fact. God has given me so much and I have returned so little by comparison that my debt to Him is infinite.

No man could have a more wonderful and loving wife and family than I. None have had so much joy and pleasure in his profession as I have. So many, here and over the country, have honored me with their friendship, concern, confidences and support that I cannot describe properly. With some of them and some of you, I have discussed relationships with God and we always seem to agree in the greatest part. If that agreement continues with you as it does with me, don't cry at my funeral. It won't be easy, maybe, for every departure leaves a wound that is slow to heal. As Red Barber said, "I'm in the catbird seat."



**Edgar W. Young, Jr., MD**
**1921 — 1987**

I'll be able to be with you, but, for a while at least, you won't be able to be with me. But God has promised us that we will all be together in His Kingdom.

Don't cry. Just wait. I love you all.

*— Edgar W. Young, Jr., MD*
*Written 1983*

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### Victor Gary Anderson, MD
### 1942 - 1987

Victor G. Anderson, MD, an emergency physician at Tulsa's St John Medical Center, died April 10 while vacationing in Florida. The Wichita native was a 1967 graduate of the University of Oklahoma College of Medicine. He returned to Wichita for his internship and residency, and from 1968 to 1971 he served as a US Army officer in Panama.

### Paul Newman Atkins, Jr., MD
### 1914 - 1987

Longtime surgeon Paul N. Atkins, Jr., MD, died April 20 in Tulsa. A native of Tulsa, Dr Atkins earned his medical degree from the University of Tennessee in 1939. During World War II he served as a lieutenant colonel and regiment surgeon with the US Army in New Guinea. He returned to Tulsa to establish his medical practice and was on the Hillcrest medical staff for more than 40 years. Dr Atkins was a Life Member of the OSMA.

### Odis A. Cook, MD
### 1905 - 1987

OSMA Life Member Odis A. Cook, MD, a general practitioner for more than 40 years, died April 4 in Oklahoma City. Born in Piedmont, Okla, Dr Cook attended the University of Oklahoma School of Medicine, where he was graduated in 1931. He first established a practice in Madill, moving later to Oklahoma City. He announced his retirement in 1979.

### Paul Lewis Nave, MD
### 1934 - 1987

Paul L. Nave, MD, an Oklahoma City ophthalmologist, died March 26, 1987. The Ponca City native was graduated from the University of Oklahoma College of Medicine in 1965. He served his internship in Florida and residency in the Canal Zone, establishing his practice in St Petersburg, Fla, in 1969. He moved to Oklahoma City in 1978.

### George Michael Willkom III, MD
### 1949 - 1987

Family practitioner George M. Willkom III, MD, of Broken Arrow died March 30, 1987. Dr Willkom earned his medical degree in 1949 at the University of Santo Tomas in the Philippines. He completed his residency training in Lubbock, Tex, in 1979 and moved to Broken Arrow to establish his practice.

### Edgar W. Young, Jr., MD
### 1921 - 1987

Edgar W. Young, Jr., MD, professor of medicine and associate dean of the University of Oklahoma College of Medicine, died in Oklahoma City on April 12. Dr Young earned his medical degree at the university in 1947. After his postgraduate training, he completed a fellowship in clinical pharmacology and spent six years with a pharmaceutical company. In 1961 he established a private practice in internal medicine in El Reno. Fifteen years later he closed that practice to assume the position of associate dean at the medical school. Dr Young also served as a member and secretary-treasurer of the Board of Medical Examiners for many years.

## IN MEMORIAM

### 1986

| | |
|---|---|
| *Marianne Elsbeth Kosbab, MD* | *June 13* |
| *William W. Rucks, Jr., MD* | *June 27* |
| *Ralph A. Smith, MD* | *July 27* |
| *Howard D. Tuttle, MD* | *August 3* |
| *Welborn W. Sanger, MD* | *September 19* |
| *William Carl Ewell, MD* | *September 20* |
| *Marcella Steel, MD* | *October 1* |
| *Terry Dwight Leming, MD* | *October 13* |
| *William Pat Fite, Jr., MD* | *October 30* |
| *Samuel Jackson McDaniel, MD* | *November 2* |
| *Iron Hawthorne Nelson, MD* | *November 12* |
| *John Robert Walter Spencer, MD* | *December 4* |

### 1987

| | |
|---|---|
| *Edward Leon Moore, MD* | *February 14* |
| *Ralph Cameron Emmott, MD* | *February 16* |
| *James Laurel Haddock, Jr., MD* | *February 19* |
| *Donald J. Blair* | *March 16* |
| *Eldon Clyde Mohler, MD* | *March 21* |
| *Paul Lewis Nave, MD* | *March 26* |
| *George Michael Willkom III, MD* | *March 30* |
| *Odis A. Cook, MD* | *April 4* |
| *Victor Gary Anderson, MD* | *April 10* |
| *Edgar W. Young, Jr., MD* | *April 12* |
| *Paul Newman Atkins, Jr., MD* | *April 20* |

**How We Live.** By Victor R. Fuchs. Cambridge, Mass: Harvard University Press, 1983. Pp 293, $17.50.

"This is a book about choices — the choices we make for ourselves, those that are made for us, and those that we make for others. It examines the choices we make in our private lives and also the ones we make collectively as citizens and voters. Through these choices — especially those concerning family, work, health, and education — we are constantly defining and redefining American society. . . ." Victor R. Fuchs, professor of economics at Stanford University, uses economics to enlarge our understanding of social problems. He is quite skilled in using economic theory to sift new meaning from routine vital statistics and routinely collected statistical data. He is equally adept at synthesis of this material, providing a thought-provoking analysis of the way we live and of the progress of Americans through their life cycle.

The facts are familiar: a doubled rate of divorce, a birth rate cut nearly in half while the percentage of illegitimate births has nearly tripled, the dismal job prospects facing the young, and an increasing dependence of many of the elderly on the federal government.

There are many subjects here of interest to physicians and on which Professor Fuchs offers interesting interpretations. These include the decline in fertility, the rise in illegitimacy, the relative stability in the proportion of low-birth-weight babies who survive, parental lack of investment in children, the sharp increases in suicide and homicide among adolescents, the increase in divorce, the conflicts between parenthood and careers, and health care for various age groups. He utilizes the latest knowledge of American life to explore three major themes — the fading family, the impact of simple demographics on individual destiny, and the effect of weighing present and future costs and benefits on individual choice.

The author ends with several specific recommendations. He concludes that there are many areas of American life that would benefit from changes in

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public policy. He gives highest priority to reform of the social security retirement system. A second area requiring attention is revamping of health insurance and concomitant changes in the organization of medical care because the cost of health care is increasing at a more rapid rate than the nation can sustain in the long run. The third area that he proposes for priority attention is increased investment in children. Efficiency suffers because many infants, children, and adolescents do not receive enough investment in human capital to realize their full potential as workers and citizens.

Professor Fuchs sets out a large number of critical issues and makes very telling interpretations of each. To be sure, some of the problems extend far beyond an economic solution or solution only by economic means; nevertheless, this is a very interesting and thought-provoking book.

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*(continued on p 396)*

# CONFIRMED

CONFIRMED BY CLINICAL EVIDENCE

ZANTAC® 150 h.s.

ranitidine HCl/Glaxo 150 mg tablets

EFFECTIVE MAINTENANCE THERAPY

for healed duodenal ulcer patients

See last page for references and
Brief Summary of Product Information.

Glaxo/ ROCHE

CONFIRMED

In two randomized, double-blind, and well-controlled clinical trials, ZANTAC 150 mg h.s. significantly superior to cimetidine 400 mg h.s. for maintenance therapy in healed duodenal ulcers.

Percent of patients with observed duodenal ulcer recurrence

| | | 0-4 months | 0-8 months | 0-12 months | No. patients |
|---|---|---|---|---|---|
| USA[1] | ranitidine 150 mg h.s. | 9% | 14%* | 16%† | 60 |
| | cimetidine 400 mg h.s. | 23% | 34% | 43% | 66 |
| UK, Ireland, Australia[2] | ranitidine 150 mg h.s. | 8%‡ | 14%‡ | 23%‡ | 243 |
| | cimetidine 400 mg h.s. | 21% | 34% | 37% | 241 |

*p=0.02
†p=0.01
‡p≤0.004
%=life-table estimates
All patients were permitted prn antacids for relief of pain.

These two trials used the currently recommended dosing regimen of cimetidine (400 mg h.s.) and ranitidine (150 mg h.s.). A comparison of other dosing regimens has not been studied.

The studied dosing regimens are not equivalent with respect to the degree and duration of acid suppression or suppression of nocturnal acid.

The superiority of ranitidine over cimetidine in these trials indicates that the dosing regimen currently recommended for cimetidine is less likely to be as successful in maintenance therapy.

Convenient once-a-night dose with a

low incidence of side effects[3]

Headache, sometimes severe, seems to be related to ranitidine administration. Other side effects have been reported; for a complete listing, see the **ADVERSE REACTIONS** section in the Brief Summary.

No significant interference with the hepatic cytochrome

P-450 enzyme system at recommended doses

ZANTAC 150 mg has no significant drug interactions with theophylline, phenytoin, or warfarin. The bioavailability of certain medications whose absorption is dependent on a low gastric pH may be altered when ZANTAC or other medications that decrease gastric acidity are administered.

**Zantac® 150**

*ranitidine HCl/Glaxo 150 mg tablets*

One tablet at bedtime
for maintenance

See next page for references and
Brief Summary of Product Information.

Glaxo/ ROCHE®

CONFIRMED

# Zantac® 150

*ranitidine HCl/Glaxo 150 mg tablets*

## *One tablet at bedtime for maintenance therapy in healed duodenal ulcer patients*


**References:**

1. Silvis SE, Griffin J, Hardin R, et al: Final report on the United States multicenter trial comparing ranitidine to cimetidine as maintenance therapy following healing of duodenal ulcer. *J Clin Gastroenterol* 1985;7(6):482-487.
2. Gough KR, Korman MG, Bardhan KD, et al: Ranitidine and cimetidine in prevention of duodenal ulcer relapse: A double-blind, randomised, multicentre, comparative trial. *Lancet* 1984;ii:659-662.
3. Data available on request, Glaxo Inc.


**ZANTAC® 150 Tablets**
**(ranitidine hydrochloride)**
**ZANTAC® 300 Tablets**
**(ranitidine hydrochloride)**

**BRIEF SUMMARY OF PRODUCT INFORMATION**

The following is a brief summary only. Before prescribing, see complete prescribing information in ZANTAC® product labeling.

**INDICATIONS AND USAGE:** ZANTAC® is indicated in:

1. Short-term treatment of **active duodenal ulcer.** Most patients heal within four weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (eg, Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within six weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD)**. Symptomatic relief commonly occurs within one or two weeks after starting therapy and is maintained throughout a six-week course of therapy.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

**CONTRAINDICATIONS:** ZANTAC® is contraindicated for patients known to have hypersensitivity to the drug.

**PRECAUTIONS:** Symptomatic response to ZANTAC® therapy does not preclude the presence of gastric malignancy.

Since ZANTAC is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see **DOSAGE AND ADMINISTRATION**). Caution should be observed in patients with hepatic dysfunction since ZANTAC is metabolized in the liver.

False-positive tests for urine protein with Multistix® may occur during ZANTAC therapy, and therefore testing with sulfosalicylic acid is recommended.

Although recommended doses of ZANTAC do not inhibit the action of cytochrome P-450 enzymes in the liver, there have been isolated reports of drug interactions which suggest that ZANTAC may affect the bioavailability of certain drugs by some mechanism as yet unidentified (eg, a pH-dependent effect on absorption or a change in volume of distribution).

Lack of experience to date precludes recommending ZANTAC for use in children or pregnant patients. Since ZANTAC is secreted in human milk, caution should be exercised when administered to a nursing mother.

**ADVERSE REACTIONS:** Headache, sometimes severe, seems to be related to ZANTAC® administration. Constipation, diarrhea, nausea/vomiting, and abdominal discomfort/pain have been reported. There have been rare reports of malaise, dizziness, somnolence, insomnia, vertigo, tachycardia, bradycardia, premature ventricular beats, and arthralgias. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients.

In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg qid IV for seven days, and in 4 of 24 subjects receiving 50 mg qid for five days. With oral administration there have been occasional reports of reversible hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice.

There have been rare reports of reversible leukopenia, granulocytopenia, thrombocytopenia, and pancytopenia.

Although controlled studies have shown no antiandrogenic activity, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving ZANTAC, but the incidence did not differ from that in the general population.

Incidents of rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia, have been reported, as well as rare cases of hypersensitivity reactions (eg, bronchospasm, fever, rash, eosinophilia) and small increases in serum creatinine.

**OVERDOSAGE:** Information concerning possible overdosage and its treatment appears in the full prescribing information.

**DOSAGE AND ADMINISTRATION**: **Active Duodenal Ulcer:** The current recommended adult oral dosage is 150 mg twice daily. An alternate dosage of 300 mg once daily at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated.

**Maintenance Therapy:** The current recommended adult oral dosage is 150 mg at bedtime.

**Pathological Hypersecretory Conditions (such as Zollinger-Ellison Syndrome):** The current recommended adult oral dosage is 150 mg twice a day. In some patients it may be necessary to administer ZANTAC 150-mg doses more frequently. Doses should be adjusted to individual patient needs, and should continue as long as clinically indicated. Doses up to 6 g/day have been employed in patients with severe disease.

**Benign Gastric Ulcer:** The current recommended adult oral dosage is 150 mg twice a day.

**GERD:** The current recommended adult oral dosage is 150 mg twice a day.

**Dosage Adjustment for Patients with Impaired Renal Function:** On the basis of experience with a group of subjects with severely impaired renal function treated with ZANTAC, the recommended dosage in patients with a creatinine clearance less than 50 ml/min is 150 mg every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosage schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

**HOW SUPPLIED:** ZANTAC® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) and unit dose packs of 100 tablets (NDC 0173-0393-47).

ZANTAC® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 tablets (NDC 0173-0344-42) and unit dose packs of 100 tablets (NDC 0173-0344-47).

**Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.**


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*diltiazem HCl/Marion*

- ***Antianginal action includes dilatation of coronary arteries, a decrease in vascular resistance/afterload, and a reduction in heart rate***
- ***Proven efficacy when used alone in angina***[1]
- ***Compatible with other antianginals***[2,3*]
- ***A safe choice for angina patients with coexisting hypertension, asthma, COPD, or PVD***[4,5]

**90 mg GREATER DOSAGE FLEXIBILITY NEW 120 mg**

***See Warnings and Precautions.**

*Please see brief summary of prescribing information on the next page.*

# CARDIZEM® diltiazem HCl/Marion

# FEW SIDE EFFECTS IN ANTIANGINAL THERAPY

## 60 mg tid or qid

**Brief Summary**
Professional Use Information

**CARDIZEM®**
(diltiazem HCl) 30 mg and 60 mg Tablets

**CONTRAINDICATIONS**

CARDIZEM is contraindicated in (1) patients with sick sinus syndrome except in the presence of a functioning ventricular pacemaker, (2) patients with second- or third-degree AV block except in the presence of a functioning ventricular pacemaker, and (3) patients with hypotension (less than 90 mm Hg systolic).

**WARNINGS**

1. **Cardiac Conduction.** CARDIZEM prolongs AV node refractory periods without significantly prolonging sinus node recovery time, except in patients with sick sinus syndrome. This effect may rarely result in abnormally slow heart rates (particularly in patients with sick sinus syndrome) or second- or third-degree AV block (six of 1,243 patients for 0.48%). Concomitant use of diltiazem with beta-blockers or digitalis may result in additive effects on cardiac conduction. A patient with Prinzmetal's angina developed periods of asystole (2 to 5 seconds) after a single dose of 60 mg of diltiazem.
2. **Congestive Heart Failure.** Although diltiazem has a negative inotropic effect in isolated animal tissue preparations, hemodynamic studies in humans with normal ventricular function have not shown a reduction in cardiac index nor consistent negative effects on contractility (dp/dt).
   Experience with the use of CARDIZEM alone or in combination with beta-blockers in patients with impaired ventricular function is very limited. Caution should be exercised when using the drug in such patients.
3. **Hypotension.** Decreases in blood pressure associated with CARDIZEM therapy may occasionally result in symptomatic hypotension.
4. **Acute Hepatic Injury.** In rare instances, significant elevations in enzymes such as alkaline phosphatase, CPK, LDH, SGOT, SGPT, and other symptoms consistent with acute hepatic injury have been noted. These reactions have been reversible upon discontinuation of drug therapy. The relationship to CARDIZEM is uncertain in most cases, but probable in some. (See PRECAUTIONS.)

**PRECAUTIONS**

**General.** CARDIZEM (diltiazem hydrochloride) is extensively metabolized by the liver and excreted by the kidneys and in bile. As with any new drug given over prolonged periods, laboratory parameters should be monitored at regular intervals. The drug should be used with caution in patients with impaired renal or hepatic function. In subacute and chronic dog and rat studies designed to produce toxicity, high doses of diltiazem were associated with hepatic damage. In special subacute hepatic studies, oral doses of 125 mg/kg and higher in rats were associated with histological changes in the liver which were reversible when the drug was discontinued. In dogs, doses of 20 mg/kg were also associated with hepatic changes; however, these changes were reversible with continued dosing.

**Drug Interaction.** Pharmacologic studies indicate that there may be additive effects in prolonging AV conduction when using beta-blockers or digitalis concomitantly with CARDIZEM. (See WARNINGS.)

Controlled and uncontrolled domestic studies suggest that concomitant use of CARDIZEM and beta-blockers or digitalis is usually well tolerated. Available data are not sufficient, however, to predict the effects of concomitant treatment, particularly in patients with left ventricular dysfunction or cardiac conduction abnormalities. In healthy volunteers, diltiazem has been shown to increase serum digoxin levels up to 20%.

**Carcinogenesis, Mutagenesis, Impairment of Fertility.** A 24-month study in rats and a 21-month study in mice showed no evidence of carcinogenicity. There was also no mutagenic response in in vitro bacterial tests. No intrinsic effect on fertility was observed in rats.

**Pregnancy.** Category C. Reproduction studies have been conducted in mice, rats, and rabbits. Administration of doses ranging from five to ten times greater (on a mg/kg basis) than the daily recommended therapeutic dose has resulted in embryo and fetal lethality. These doses, in some studies, have been reported to cause skeletal abnormalities. In the perinatal/postnatal studies, there was some reduction in early individual pup weights and survival rates. There was an increased incidence of stillbirths at doses of 20 times the human dose or greater.

There are no well-controlled studies in pregnant women, therefore, use CARDIZEM in pregnant women only if the potential benefit justifies the potential risk to the fetus.

**Nursing Mothers.** Diltiazem is excreted in human milk. One report suggests that concentrations in breast milk may approximate serum levels. If use of CARDIZEM is deemed essential, an alternative method of infant feeding should be instituted.

**Pediatric Use.** Safety and effectiveness in children have not been established.

**ADVERSE REACTIONS**

Serious adverse reactions have been rare in studies carried out to date, but it should be recognized that patients with impaired ventricular function and cardiac conduction abnormalities have usually been excluded.

In domestic placebo-controlled trials, the incidence of adverse reactions reported during CARDIZEM therapy was not greater than that reported during placebo therapy.

The following represent occurrences observed in clinical studies which can be at least reasonably associated with the pharmacology of calcium influx inhibition. In many cases, the relationship to CARDIZEM has not been established. The most common occurrences as well as their frequency of presentation are: edema (2.4%), headache (2.1%), nausea (1.9%), dizziness (1.5%), rash (1.3%), asthenia (1.2%). In addition, the following events were reported infrequently (less than 1%):

| | |
|---|---|
| Cardiovascular: | Angina, arrhythmia, AV block (first degree), AV block (second or third degree — see conduction warning), bradycardia, congestive heart failure, flushing, hypotension, palpitations, syncope. |
| Nervous System: | Amnesia, gait abnormality, hallucinations, insomnia, nervousness, paresthesia, personality change, somnolence, tinnitus, tremor. |
| Gastrointestinal: | Anorexia, constipation, diarrhea, dysgeusia, dyspepsia, mild elevations of alkaline phosphatase, SGOT, SGPT, and LDH (see hepatic warnings), vomiting, weight increase. |
| Dermatologic: | Petechiae, pruritus, photosensitivity, urticaria. |
| Other: | Amblyopia, dyspnea, epistaxis, eye irritation, hyperglycemia, nasal congestion, nocturia, osteoarticular pain, polyuria, sexual difficulties. |

The following postmarketing events have been reported infrequently in patients receiving CARDIZEM: alopecia, gingival hyperplasia, erythema multiforme, and leukopenia. However, a definitive cause and effect between these events and CARDIZEM therapy is yet to be established.

*Issued 7/86*

See complete Professional Use Information before prescribing.

**References: 1.** Pepine CJ, Feldman RL, Hill JA, et al: Clinical outcome after treatment of rest angina with calcium blockers: Comparative experience during the initial year of therapy with diltiazem, nifedipine, and verapamil. Am Heart J 1983, 106(6):1341-1347. **2.** Shapiro W: Calcium channel blockers: Actions on the heart and uses in ischemic heart disease. Consultant 1984,24(Dec):150-159. **3.** Johnston DL, Lesowoy R, Human DP, et al: Clinical and hemodynamic evaluation of propranolol in combination with verapamil, nifedipine and diltiazem in exertional angina pectoris: A placebo-controlled, double-blind, randomized, crossover study. Am J Cardiol 1985,55:680-687. **4.** Cohn PF, Braunwald E: Chronic ischemic heart disease, in Braunwald E (ed): Heart Disease: A Textbook of Cardiovascular Medicine, ed 2. Philadelphia, WB Saunders Co, 1984, chap 39. **5.** Schroeder JS: Calcium and beta blockers in ischemic heart disease: When to use which. Mod Med 1982,50(Sept):94-116.

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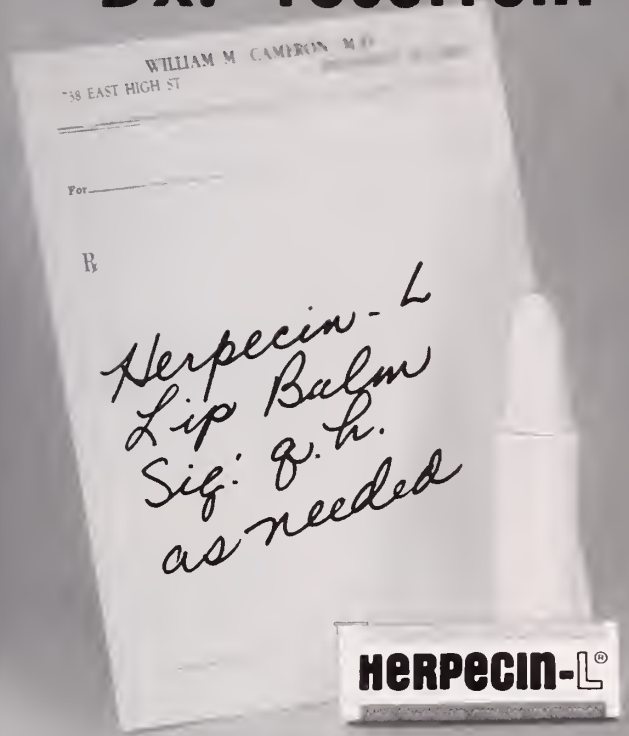


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**LIBRIUM® Ⓥ**
**chlordiozepoxide HCl/Roche**
**5-mg, 10-mg, 25-mg capsules**
**Before prescribing, please consult complete product information, a summary of which follows:**
**Indications:** Management of anxiety disorders; short-term relief of anxiety symptoms, acute alcohol withdrawal symptoms, preoperative apprehension and anxiety. Usually not required for anxiety or tension associated with stress of everyday life. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.
**Contraindications:** Known hypersensitivity to the drug.
**Warnings:** Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms (including convulsions) reported after abrupt cessation of extended use of excessive doses are similar to those seen with barbiturates. Milder symptoms reported infrequently when continuous therapy is abruptly ended. Avoid abrupt discontinuation; gradually taper dosage.
***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
**Precautions:** In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically. Due to isolated reports of exacerbation, use with caution in patients with porphyria.
**Adverse Reactions:** Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
**Usual Daily Dosage:** Individualize for maximum beneficial effects. *Oral—Adults* Mild and moderate anxiety disorders and symptoms, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)
**Supplied:** Librium® (chlordiazepoxide HCl/Roche) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in boxes of 4 reverse-numbered cards of 25, and in boxes containing 10 strips of 10. Libritabs® (chlordiazepoxide/Roche) *Tablets*, 5 mg and 10 mg—bottles of 100 and 500; 25 mg—bottles of 100. With respect to clinical activity, capsules and tablets are indistinguishable. PI 0286

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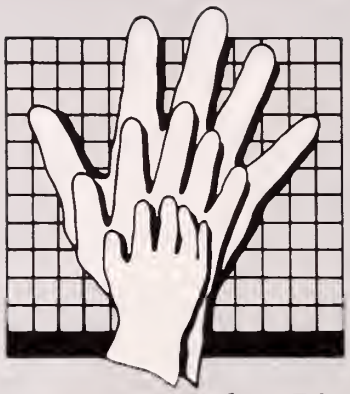

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**News**

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■ ***Medicine: Playing Politics for the Future*** **is the** title of a videotape now available from the American Medical Political Action Committee (AMPAC). The tape, which features comments from Norman L. Dunitz, MD, immediate past president of the OSMA, explains how the physician can affect the political process. Those wishing to see the program should contact OSMA Associate Director Robert W. Baker III. The videotape also will be distributed nationally by the AMA.

■ **A national conference on impaired physicians** and other health professionals will be hosted by the American Medical Association (AMA) in Chicago October 8-11, 1987. Eighth in a series of AMA conferences on physician impairment, the meeting will focus on the need for increased collaborative efforts among health professions in educating themselves, state licensure boards, and medical educators. Registration information can be obtained from Janice Robertson, (312) 645-5079.

■ **State physicians and their colleagues, em**ployees, and spouses are reminded that the JOURNAL counts on *you* for state news and other items of interest. We want to know about physicians' achievements and awards, involvement in community projects, hobbies and favorite pastimes. We also encourage the busy practitioner to pass along that anecdote, observation, or case report thought too brief or insignificant to write up. If it means something to you, it could mean something to others, too. Don't write off the JOURNAL. Write *for* the JOURNAL and *to* the JOURNAL.

■ **Elvin M. Amen, MD, Bartlesville, has been** elected to a three-year term as advisor to the Oklahoma Medical Assistants Society. Dr Amen was president of the OSMA in 1985-86.

■ **The Young Physicians Section of the Okla**homa State Medical Association became a functioning entity at the OSMA's Annual Meeting in Oklahoma City last month. The new section is open to physicians under 40 years of age or in their first five years of practice. (Approximately 40% of the physicians in the US today are under age 40; 20% of them are women.) Doctors interested in the new section should contact OSMA Associate Director Robert W. Baker III.

■ **John W. Records, MD, Oklahoma City, is the** recipient of the Oklahoma City Clinic's first Alexander Poston Leadership Award. The award was established in memory of Alexander Poston, MD, former president of the clinic's board of directors, who died in 1986 at the age of 47. Dr Records, who retired last summer, was given the award in recognition of his more than 45 years of service and leadership at the clinic.

■ **As many as 56% of all smokers say they** have never been advised by a physician to quit smoking. The finding, published in the *Journal of the American Medical Association,* is based on two random statewide surveys of 5,875 Michigan adults, conducted in 1980 and 1983. "Of smokers who had seen a physician in the previous year, only 44% reported that they had ever been told to quit smoking by a physician," say Robert F. Anda, MD, MS, of the Michigan Department of Public Health, and colleagues. Smokers who had experienced a heart attack or stroke were more likely to have received such advice, but smokers with additional risk factors (hypertension, obesity, diabetes, sedentary lifestyle, or oral contraceptive use) were no more likely to have been told to quit than other smokers. Blacks and young men were less likely to have been told to quit than other groups.

■ **The Heimlich maneuver can save people from** choking but also can have side effects, reports of which include pharyngeal abrasion, esophageal laceration, retinal detachment and fractured ribs. A report in May's *Archives of Pathology and Laboratory Medicine* describes a new apparent complication — a ruptured prosthetic aortic valve. Cary S. Passik, MD, of the Mayo Clinic, Rochester, Minn, and colleagues say the problem involved a 74-year-old woman whose son performed the Heimlich maneuver on her during a choking episode three years after the valve surgery. The woman later developed symptoms of aortic insufficiency and needed further surgery. Examination of the original valve revealed a perforation and a tear, the report says, presumably due to a jump in intrathoracic pressure after the Heimlich maneuver. "Patients with unexplained aortic prosthetic insufficiency should be questioned as to whether the Heimlich maneuver has been previously performed," it concludes.

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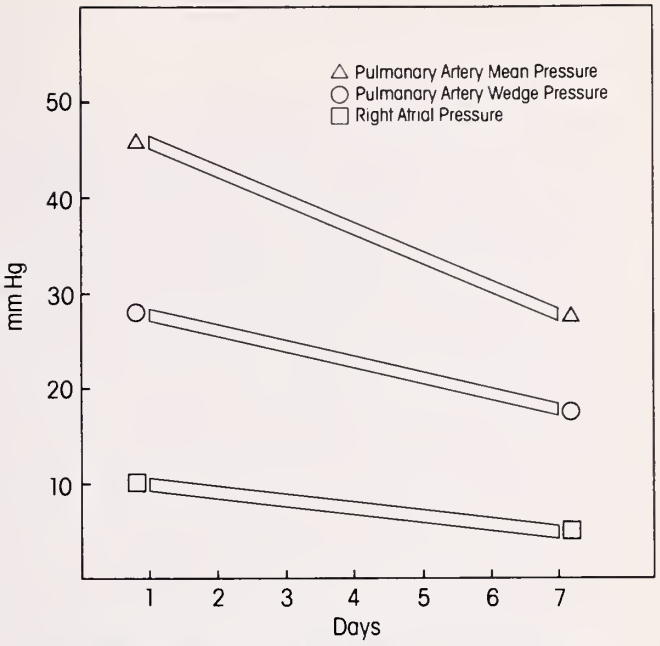


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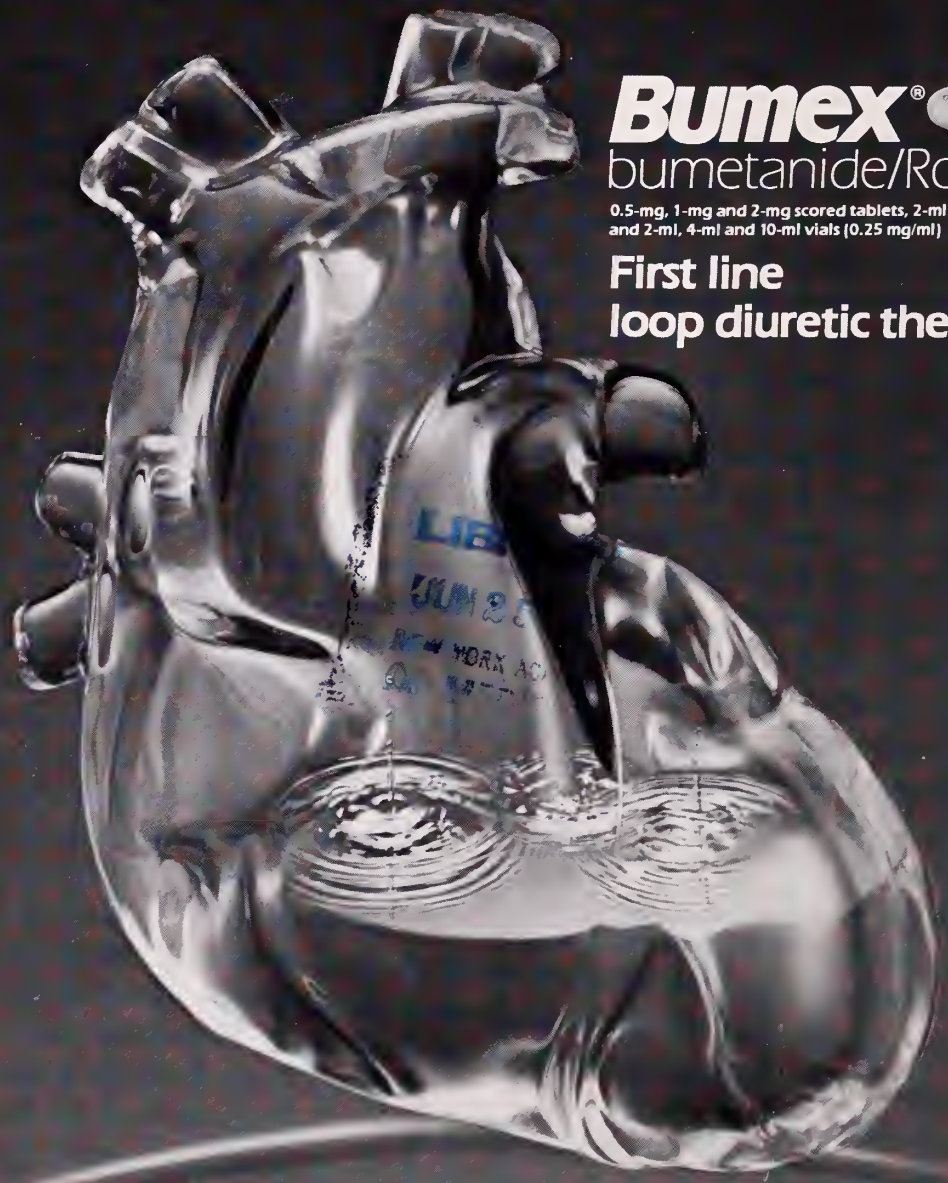

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